Journal of Population Economics (2025) 38:67
https://doi.org/10.1007/s00148-025-01124-0

ORIGINAL PAPER

Male excess mortality during the epidemiological transition: theory and evidence from India

Astrid Krenz[1,2] · **Holger Strulik**[3]

Received: 4 January 2025 / Accepted: 22 August 2025

Abstract
At any given age, adult men die at a higher rate than women. In many developed countries, increasing excess mortality of men has been demonstrated for cohorts born in the late nineteenth century and thereafter. The decline in infectious diseases is believed to have contributed to the increase in male excess mortality. Here, we focus on India during 1990–2019, a period in which the Indian states experienced, to varying degrees, the epidemiological transition. We show that male excess mortality evolves positively over the observation period, is greater in later-born cohorts, and is strongly associated with the decline in infectious disease mortality. We propose a simple theory that explains these facts by a greater influence of infections on the biological aging of women compared to men. We calibrate the model with Indian data and show that it can replicate the feature of rising male excess mortality over time and birth year of cohorts.

Keywords Male excess mortality · Epidemiological transition · Biological aging · India

JEL Classification I15 · J11 · J16 · N35

Responsible editor: Oded Galor

✉ Holger Strulik
holger.strulik@wiwi.uni-goettingen.de

Astrid Krenz
a.krenz@lse.ac.uk

1 London School of Economics and Political Science, Canada Blanch Centre & Data Science Institute, Houghton Street, London WC2A 2AE, UK

2 Department of Management and Economics, Center for Entrepreneurship, Innovation and Transformation (CEIT), Ruhr University Bochum, Universitaetsstrasse 150, 44801 Bochum, Germany

3 Department of Economics, University of Goettingen, Platz der Goettinger Sieben 3, 37073 Goettingen, Germany

Published online: 09 September 2025

1 Introduction

Women today live significantly longer than men in almost all countries worldwide. This phenomenon, however, is relatively recent. In developed countries, excess mortality of adult men emerged in cohorts born in the late nineteenth century and then increased substantially for about half a century before stabilizing in some regions (Beltran-Sanchez et al. 2015; Goldin and Lleras-Muney 2019). The female-male difference in life expectancy at age 45 has continued to rise in most developing countries, reaching approximately 4 years in Asia, Latin America, and the world average. In Europe, it reached a maximum difference of 6.5 years in the early 1990s and has since modestly declined to 5.7 years. India represents a latecomer to this trend, with the gender gap in life expectancy at age 45 increasing from 0.9 years in 1950 to 2.5 years in 2023 (UN 2024).

This paper investigates the dynamics of male excess mortality during the epidemiological transition in India from 1990 to 2019. Specifically, we define male excess mortality as the ratio of adult male-to-female mortality and focus on how the shifting burden from infectious to non-communicable diseases (NCDs) affects gender differences in mortality. Our analysis extends the existing literature by examining a developing country in the midst of an epidemiological transition, rather than retrospectively analyzing data from already-developed nations. India is also characterized by an infectious disease burden that differs from the previously studied Western countries and that varies substantially across the Indian states and over time, a feature that we leverage in our panel and cohort analysis.

We propose and test a theoretical framework centered on the "inflammaging" pathway — the process by which lifetime exposure to infections contributes to chronic inflammation that accelerates biological aging. We hypothesize that women's stronger inflammatory responses to infections increase their vulnerability to chronic diseases later in life. As infectious disease burden declines during economic development, women consequently benefit more than men in terms of reduced mortality from both infectious diseases and NCDs, leading to increasing male excess mortality.

The literature offers both biological and behavioral explanations for women's longevity advantage. Biologically, women tend to develop more acute illnesses and non-fatal chronic conditions, while men are more prone to life-threatening chronic diseases such as coronary heart disease, certain cancers, cerebrovascular disease, and liver and kidney diseases (Verbrugge 1985; Rieker and Bird 2005; Case and Paxson 2005). Hormonal factors (such as estrogen's protective effect against cardiovascular disease), genetic advantages (including protection against X-linked disorders), and autoimmune differences contribute to this disparity (Holden 1987; Austad 2006; Oksuzyan et al. 2008). Behaviorally, women generally engage in fewer risk-taking health behaviors like smoking and drinking, and often maintain healthier diets with more fruits and less meat and salt (Waldron 1993; Wardle et al. 2004).

Relatively few studies have examined the dynamics of male excess mortality. Why did it first emerge in generations born in the late nineteenth century? Why did it then increase almost uniformly in developed countries? And why do developing countries experience similar dynamics, but with a delay in the onset of the process and at

different speeds? A potential explanation is that economic development triggered behavioral changes that emerged earlier or more strongly in men, such as increased smoking and meat consumption. Beltran-Sanchez et al. (2015) found that in their sample of 13 developed countries, smoking can account for about 30% of the increase in excess male mortality and suggest that the remaining increase may be attributable to declining infection-driven inflammation. This view is further corroborated by Goldin and Lleras-Muney (2019) by showing that in the US deaths from infectious diseases began to decline markedly at the end of the nineteenth century and that young females died from infectious at higher rates than males. Women thus benefited more from the decline in infectious diseases than men, and their advantage increased as the epidemiological transition progressed due to the lower lifetime exposure to infections of later-born cohorts.

The concept of inflammaging helps to explain this pattern. Inflammaging refers to the progressive development of a chronic pro-inflammatory state resulting from lifetime exposure to pathogens. This chronic inflammation contributes to various age-related diseases, including cardiovascular disease, diabetes, cancers, and neurodegenerative disorders (Franceschi and Campisi 2014; Bektas et al. 2018; Ferrucci and Fabbri 2018; Franceschi et al. 2018). Particularly, after puberty, the inflammatory responses to infections are much stronger in women (Klein and Flanagan 2016). Strong inflammatory responses lead to better protection against (death from) infections but also to stronger inflammaging and therewith to higher prevalence of chronic diseases in old age. Among older Americans, where there was likely little sex difference in disease exposure during childhood and young adulthood, women showed significantly worse indices of inflammatory biomarkers (Yang and Kozlosk 2011; see also Crimmins et al. 2019). Several studies have demonstrated how individual infection histories contribute to the development of chronic health deficits (Finch and Crimmins 2004; Finch 2010; Santoro et al. 2021). Costa (2000; 2002) showed that chronic diseases among older American men decreased by over 60% from the early nineteenth century to the 1970s, with a significant portion attributable to declining infections.

To measure chronic, aging-related health deficits, we utilize the frailty index approach developed by Mitnitski et al. (2001), which assesses an individual's health as the proportion of aging-related health deficits present from a comprehensive list of potential deficits. The frailty index has proven to be an excellent predictor of mortality (Rockwood and Mitnitski 2007; Blodgett et al. 2016; Hosseini et al. 2022). The association between the frailty index and the mortality rate has been precisely estimated as a log-log association (a power law) with a sex-specific exponent around 3 (Mitnitski et al. 2002a, b; Dalgaard et al. 2022). Another robust result of previous studies is an exponential increase of the frailty index with age at a rate of around 3% per year (Mitnitski and Rockwood 2016; Abeliansky and Strulik 2018; Abeliansky et al. 2020; Dalgaard et al. 2022).

Previous studies on biological aging in India include work by Chatterji et al. (2008), Bloom et al. (2014, 2021), Patel et al. (2021), and Chaudhary and Arokiasamy (2019). Particularly relevant is Dandona et al.'s (2017) approach to measuring the epidemiological transition by comparing disability-adjusted life years (DALYs) for communicable and non-communicable diseases. Canudas-Romo et al. (2015)

investigated sex differentials in life expectancy across Indian states from 1970 to 2013, finding women had an advantage in adult life expectancy.

Our paper makes several contributions to this literature. First, we use data from the Global Burden of Disease Study for India (GBD-I) from 1990 to 2019 and compute male excess mortality for the Indian population across states, years, and age groups. We exploit the panel structure of the GBD-I data to construct cohorts and explore male excess mortality for Indian cohorts born 1895–1990. Following Dandona et al. (2017), we compute the epidemiological transition ratio, defined as the ratio of all-age DALYs due to communicable, maternal, neonatal, and nutritional diseases divided by all-age DALYs due to non-communicable diseases. We then show that male excess mortality rises with the progression of the epidemiological transition, both across and within Indian states.

Second, we develop a theoretical model of health deficit accumulation based on Dalgaard and Strulik (2014) that incorporates the inflammaging pathway. The model predicts that as a society undergoes an epidemiological transition with declining infectious disease burden, male excess mortality from NCDs will increase if women experience stronger inflammatory responses to infections.

Third, we calibrate our theoretical model with Indian data and demonstrate that it accurately predicts increasing male excess mortality from NCDs during the epidemiological transition. The model also captures the broad associations between male excess mortality and the level of the epidemiological transition across age groups, birth cohorts, and the Indian states. Finally, we use the model for an exploratory computational history by predicting ex-post male excess mortality for cohorts born between 1870 and 1970.

It should be noted that our analysis focuses on one particular mechanism of male excess mortality. Applying Occam's razor, we do not to provide a comprehensive explanation of all underlying causes. In particular, we do not account for gender differences in health behaviors. For instance, increasing rates of smoking and alcohol consumption among men relative to women in the birth cohorts studied may have also played a role in the rise of male excess mortality. The limitations of our approach are further discussed in the Discussion and Conclusion section.

2 The epidemiological transition and male excess mortality in India

2.1 Data and sampling

We use data from the Global Burden of Disease study for India (Indian Council of Medical Research, 2017), henceforth cited as GBD-I. The data are sourced from IHME (2021). The database covers the 29 Indian states and the Union Territory of Delhi (henceforth, cited as states). The data are available for the period 1990 to 2021, but we focus on the period 1990–2019 to obtain long-term trends of the epidemiological transition that are not affected by the recent shock of the COVID pandemic.

The age group by year structure of the GBD-I data allows for the construction of cohorts. Because we focus on the excess mortality of adult men, we included only

adults aged 20 and above. We kept every fifth year of observation and constructed 20 cohorts. The youngest considered cohort is 20–24 years old in 2019 and thus born 1995–1999 (addressed as vintage 1995). The oldest cohort is 90–94 years old in 1990 (vintage 1900).

From the GBD-I database, we collected data by age group, year, and state on death rates from communicable diseases, non-communicable diseases, and all causes, as well as prevalence rates of 33 aging-related non-communicable diseases. Additionally, we collected data at the year-by-state level on disability-adjusted life years (DALYs) measured in DALYs per 1000 persons. Following Dandona et al. (2017), we used these data to compute the epidemiological transition ratio (ETR), defined as the ratio of all-age DALYs due to communicable, maternal, neonatal, and nutritional diseases divided by all-age DALYs due to non-communicable diseases. The ETR is our main indicator of the epidemiological transition. Dandona et al. (2017) also consider the epidemiological transition level (ETL). A high ETL value indicates a well-advanced epidemiological transition, and it is therefore associated with a *low* ETR value.[1]

Furthermore, we used the GBD-I database to construct the frailty index for the Indian population stratified by state, year, sex, and age. As discussed in the Introduction, the frailty index is a widely used measure for individual aging in gerontology and the medical sciences (Howlett et al. 2021). It is constructed as the proportion of the total potential deficits that an individual has. The criteria for the selection of health deficits as items of the index are outlined in Searle et al. (2008): they need to be aging-related (prevalence increasing in age), associated with health status, not saturate too early, and cover a broad range of deficits. Here, we follow Dalgaard et al. (2022) and compute the frailty index for populations. Specifically, the average frailty index of people in age group a, gender g, from Indian state s is computed as $D_{ags} = \frac{1}{n}\sum_{d=1}^{n}\frac{P_{dags}}{P_{ags}}$, that is as the sum of n prevalence rates of age-related conditions d in age group a, gender g, and state s. We arrived at 33 health deficits (prevalence rates) that are listed in the Supplementary Material.

2.2 The epidemiological transition

During our observation period, India experienced a massive decline in mortality from communicable diseases. The panel on the left-hand side of Fig. 1 shows age-standardized death rates for men (blue squares) and women (red circles). While female mortality declined somewhat more quickly, the common trend for both sexes can be accurately described by a decline at a constant rate of 3.5% per year (black line). An alternative and perhaps more appropriate description of the epidemiological transition can be obtained by comparing communicable with non-communicable diseases. The center panel of Fig. 1 shows the epidemiological transition ratio (ETR) computed

[1] IHME (2021) reports aggregate deaths and DALYs from communicable diseases together with maternal, neonatal, and nutritional diseases in one variable, which is thus a biased measure of communicable diseases. However, for our study, the bias is limited because we focus on adults, and in the benchmark calibration, we focus on people over 45 years old, i.e., on age groups for which deaths from maternal, neonatal, and nutritional diseases play an insignificant role.

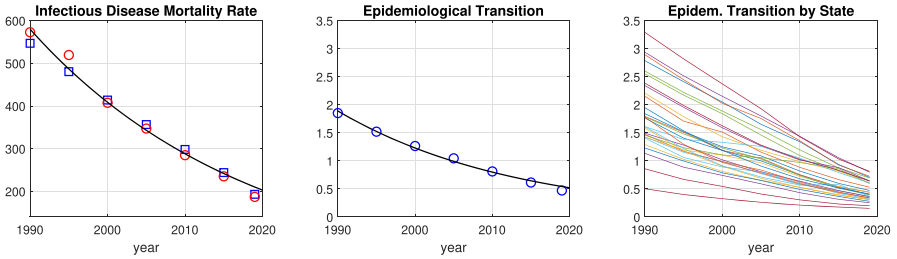

Fig. 1 The epidemiological transition in India. Left: communicable disease death rate per 100,000 (age-standardized) for men (blue squares) and women (red circles). The fitted trend declines at a rate of 3.5% per year (black line). Center: epidemiological transition ratio (Dandona et al. 2017): all-age DALYs due to communicable diseases divided by all-age DALY due to non-communicable diseases, India average. The fitted trend declines at a rate of 4.3% per year (black line). Right: epidemiological transition ratio by state

as the ratio of all-age DALYs due to communicable, maternal, neonatal, and nutritional diseases divided by all-age DALYs due to non-communicable diseases. The (unweighted) average ETR of all states fell from a value of slightly less than 2 in 1990 to a value of slightly less than 0.5 in 2019. The decline can be well approximated with a constant trend at a rate of 4.3.

The aggregate trend hides a great variety in the level of the epidemiological transition across India. The panel on the right-hand side of Fig. 1 shows the development of the ETR for the 30 Indian states, which ranges from forerunner state Kerala, where the ETR declined from 0.50 in 1990 to 0.15 in 2019, to latecomer state Uttar Pradesh, where the ETR declined from 3.29 in 1990 to 0.89 in 2019. Despite the great difference in levels, the most striking impression from the figure is the great similarity in the trend of decline (very little overtaking took place). With some abstraction, one could say that the latecomer states are on the same path as the forerunner states, but only arrive at a certain level of epidemiological transition later. This is a view we will take when calibrating the model.

2.3 Male excess mortality

As explained in the Introduction, the related literature proposes an inflammaging channel for rising male excess mortality, which is based on the insight that greater inflammatory responses to infections cause an earlier onset of chronic, aging-related diseases and earlier death from these diseases. If women are more exposed to infectious diseases than men or if they are subject to greater inflammation responses to infections, they will benefit more from a decline of infectious diseases than men in terms of lower death rates from chronic (i.e., non-communicable) diseases. If women have a survival advantage in an infection-free environment, the decline in infectious diseases leads to increasing male excess mortality from chronic diseases. Our main dependent variable is thus the ratio of male-to-female mortality from non-communicable diseases (NCD mortality).

The panel on the left-hand side of Fig. 2 shows that the average male NCD mortality across all states and age groups exceeded female NCD mortality by a factor of 1.3 at

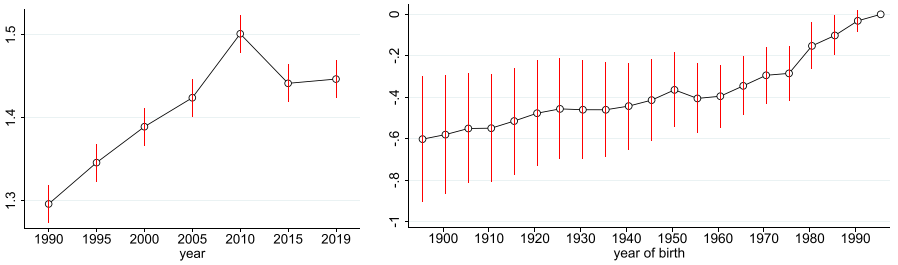

Fig. 2 Trends of male excess NCD mortality by year and cohort. Left: estimates from regression of male excess NCD mortality on a full set of year dummies (omitting the regression constant), along with their 95% confidence bands. Right: estimates from regression of male excess NCD mortality on cohort dummies (indicated by birth years 1900, 1905, 1910,...), along with their 95% confidence bands. The regression controls for state and age fixed effects. Standard errors are clustered at the state level

the beginning of the observation period and increased to about a factor of 1.45 at the end of the observation period.

The panel on the right-hand side of Fig. 2 shows how excess mortality evolved across cohorts within states. It depicts the estimation results from a regression of male excess NCD mortality on cohort dummies (indicated by birth years 1900, 1905, 1910,...), controlling for state and age fixed effects. By absorbing age fixed effects, we calculate male excess mortality for the same age group across different cohorts. The estimates reveal a strong increase of excess mortality over the last century. The point estimate for the 1900 cohort suggests that excess mortality increased by 0.6 over the observation period, which — based on its average level of 1.45 in 2020 — means that Indian men born early in the twentieth century had a survival advantage. Since later-born cohorts experienced a lower level of the epidemiological transition over their lifetime, the cohort diagram indirectly supports the inflammaging channel of increasing male excess mortality.

A potential concern with the cohort analysis is that not all cohorts are observed at all ages. To address this, the Supplementary Material presents male excess mortality at ages 45 and 65 for all cohorts that are continuously observed in the GBD-I data — specifically, cohorts born between 1945 and 1975 (age 45) and between 1925 and 1955 (age 65), as shown in Fig. A.1. The patterns observed in Fig. 2 are maintained: excess NCD mortality increases with later birth cohorts, and the magnitude of the increase remains comparable to that in Fig. 2 when the observation window is restricted to the cohorts shown in Fig. A.1.

For additional comparison, we also computed male excess mortality from all causes at ages 45 and 65 (Fig. A.2), which similarly rises across birth cohorts. Over a 30-year span, the increase is about 0.2 for 45-year-old men and around 0.1 for those aged 65. To further extend the analysis, we drew on Indian census data from HLD (2025) to compute male excess mortality at these ages over a wider cohort range. Figure A.3 displays results for cohorts born between 1925–1971 (age 45) and 1905–1951 (age 65). Again, we see the upward trend in excess mortality. The size of the increase is comparable to that observed in the GBD-I data, lending additional external validation to our main findings.

Figure 3 shows how male excess mortality varied across the Indian states in the year 2015. It depicts the state fixed effects obtained from a regression of male excess NCD mortality on state and age fixed effects. The figure plots these coefficients against the state-specific ETR in 2015. On average, male excess mortality is higher in states where the epidemiological transition is more advanced (i.e., where the ETR is low). Moving from Bihar (ETR $\approx$ 1.1) to Goa (ETR $\approx$ 0.3) leads to an increase of male excess mortality by about 0.25.

Following the seminal study of Dandona et al. (2017), our preferred measure of the epidemiological transition is the ETR. However, a potential concern could be the partial endogeneity of the ETR since mortality from non-communicable diseases (used to compute male excess mortality) is also included in the denominator of the ETR. To eliminate this concern, we show the robustness of results when the ETR is replaced by age-standardized mortality from communicable diseases for females (CD mort^F) and males (CD mort^M). Data for these alternative measures of the epidemiological transition were also sourced from IHME (2021). The raw correlation between the ETR and CD mort^F is 0.90, and the partial correlation, controlling for state fixed effects, is 0.95. Figure A.4 in the Supplementary Material shows the partial correlation between the ETR and CD mort^F on the left-hand side and the partial correlation between CD mort^F and males CD mort^M on the right-hand side. The fact that these measures are highly correlated eliminates the concern that the evolution of the ETR could be dominated by improvements in non-communicable disease mortality. Finally, Fig. A.5 in

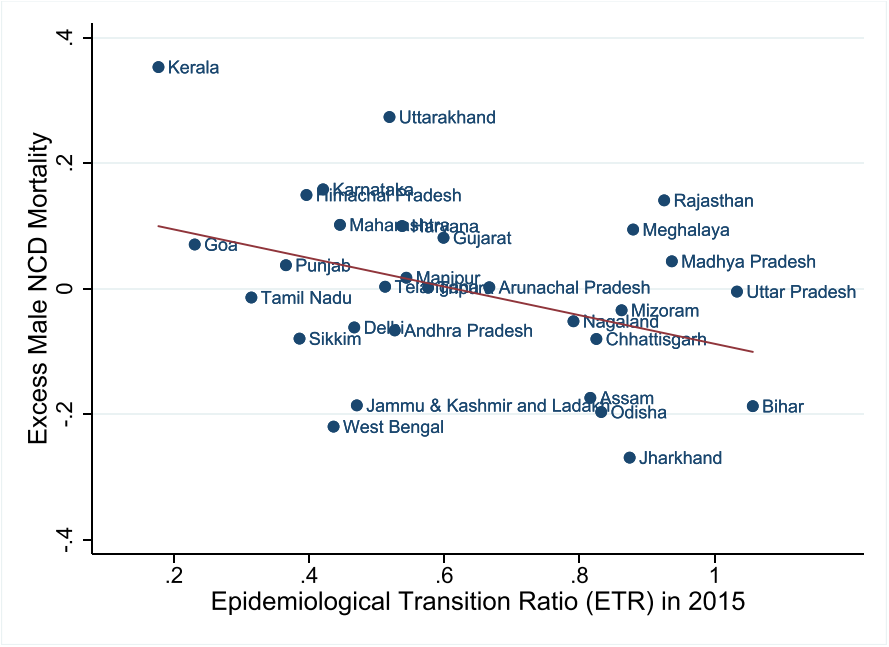

Fig. 3 Epidemiological transition ratio and male excess NCD mortality ratio by state. The figure shows the state fixed effects from a regression of male excess NCD mortality on state and age fixed effects against the state-specific ETR value in 2015. The regression line is $0.140 - 0.228 ETR$

the Supplementary Material replicates Fig. 3, but replaces the epidemiological transition ratio (ETR) with the age-standardized female mortality from communicable diseases. Although some differences between the two figures are noticeable, the overall impression is that the variation in the ETR across states and over time within states can be equally well captured by the variation in infectious disease mortality for either men or women.

Finally, we assess the quantitative association between the epidemiological transition and male excess mortality with OLS regressions. In Table 1, Column (1) shows the results of a pooled regression of male excess NCD mortality on ETR, age, and age squared. By inserting the average decline of ETR from about 2 to about 0.5 (see center panel Fig. 1) into the point estimate from column (1), we obtain a predicted increase of male excess NCD mortality by 0.165, which is about the increase observed in Fig. 2 (left). Additionally, excess mortality is potentially hump-shaped in age. However, when we feed age into the regression, we obtain a peak at age 6, implying that excess mortality has a negative age gradient throughout the adult life.

Column (2) shows results when controlling for state fixed effects. The robustness of the ETR coefficient in the within-state estimates is indicative of a similar exponential decline of ETR across states. To see this, notice that for a constant rate of ETR decline, the contemporaneous value of ETR is informative about the ex-ante course of the epidemiological transition. For a given age (controlling for age), people then have experienced the same proportional decline of ETR irrespective of the vastly varying degrees of ETR across states (see Fig. 1).

In columns (3) and (4), we replicate these regressions with all-cause excess mortality of men as the dependent variable. While the age coefficients remain stable, the ETR coefficients double. This means that mortality from infectious diseases fell more in women than in men during the epidemiological transition, which in turn supports the idea that women suffered more from infectious disease mortality due to higher exposure during the epidemiological transition.

In columns (5) and (6), we use female infectious disease mortality as an alternative indicator of the epidemiological transition. Once again, the association with male excess mortality is significantly negative, and the size of the ETR coefficient decreases slightly after controlling for state fixed effects. The estimates indicate that a reduction in female infectious disease mortality by 3 per 1000 (approximately the difference between Uttar Pradesh and Goa) is associated with an increase of male excess mortality from non-communicable diseases by about 12 percentage points.

Columns (7) and (8) present results using the male infectious disease mortality rate as the measure of the epidemiological transition. The association with male excess mortality from non-communicable diseases remains significantly negative, and the size of the coefficient is only slightly smaller than in columns (5) and (6). The similarity of results, regardless of whether male or female infectious disease mortality is used as the explanatory variable, suggests that the rise in male excess mortality is unlikely to be driven by gender-biased selection or survivor effects — that is, it is not merely due to a larger number of less healthy men surviving into old age as infection rates decline. If this were the case, we would expect a noticeably larger coefficient in the models using male infectious disease mortality, which is clearly not the case.

Table 1 Male excess mortality and the epidemiological transition

	Ex NCD (1)	Ex NCD (2)	Ex total (3)	Ex total (4)	Ex NCD (5)	Ex NCD (6)	Ex NCD (7)	Ex NCD (8)
ETR	−0.110***	−0.099***	−0.209***	−0.201***				
	(0.030)	(0.028)	(0.033)	(0.029)				
CD mort^F					−0.042***	−0.038***		
					(0.010)	(0.010)		
CD mort^M							−0.038***	−0.037***
							(0.011)	(0.012)
Age	0.086***	0.086***	0.101***	0.101***	0.086***	0.086***	0.086***	0.086***
	(0.015)	(0.015)	(0.009)	(0.009)	(0.015)	(0.015)	(0.015)	(0.015)
Age^2	−0.007***	−0.007***	−0.008***	−0.008***	−0.007***	−0.007***	−0.007***	−0.007***
	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)
Constant	1.417***	1.405***	1.462***	1.453***	1.451***	1.435***	1.435***	1.431***
	(0.072)	(0.068)	(0.064)	(0.057)	(0.069)	(0.072)	(0.070)	(0.077)
Observations	6,300	6,300	6,300	6,300	3,150	3,150	3,150	3,150
R^2	0.259	0.402	0.353	0.467	0.270	0.406	0.257	0.400
State FE	No	Yes	No	Yes	No	Yes	No	Yes

This table reports OLS estimates in regressions where the dependent variable is male excess NCD mortality (Ex NCD) in columns (1) and (2) and (5) to (8) and male excess all-cause mortality (Ex total) in columns (3) and (4). Independent variables are the epidemiological transition ratio (ETR) in columns (1) to (4), the age-standardized mortality rate from communicable diseases for females (CD mort^F) in columns (5) and (6), and the age-standardized mortality rate from communicable diseases for males (CD mort^M) in columns (7) and (8). All regression control for age and age squared. Standard errors (reported in parentheses) are robust and clustered at the state level. *$p < 0.1$; **$p < 0.05$; ***$p < 0.01$

3 The epidemiological transition and aging of men and women: theory

Following the health deficit model (Dalgaard and Strulik 2014), we conceptualize human aging as the self-productive accumulation of health deficits $D_j(t, v)$, in which $j = F, M$ indicates sex (female, male), t is age, and v is the cohort (or vintage) of the individual. Existing health deficits are conducive to the development of new health deficits at a rate μ_j. Additionally, exposure to an infectious disease environment also promotes the development of new health deficits, such that new health deficits evolve as

$$\frac{\mathrm{d}D_j(t,v)}{\mathrm{d}t} = \mu_j D_j(t,v) + \alpha_j E(t,v), \tag{1}$$

in which μ_j is the sex-specific force of aging and α_j is the sex-specific inflammation coefficient. $E(t, v)$ is the disease environment faced by cohort v at age t. We assume that both men and women face the same disease environment. If women are more exposed to infections, it would be reflected in a larger inflammation coefficient α_F. The model, therefore, does not distinguish whether greater inflammation is due to greater exposure to infections or due to greater immune system response to infections. In other words, the model is agnostic of whether differences α_j are gender-based (driven by the environment) or sex-based (driven by biology).

The disease environment is measured by the epidemiological transition ratio (ETR) and it declines at a constant rate β, as motivated in Section 2. The initial ETR at the time of birth of cohort v is denoted by $e_0(v)$. The assumption of a constant rate of decline of the ETR is a simplifying assumption that allows us to solve the problem analytically. The assumption is empirically justified by the approximately constant rate of infectious disease environment decline observed in Fig. 1. Notice that this modeling setup does not imply that exposure to diseases is constant across birth cohorts. Later born cohorts (indexed by larger v) face a lower initial disease environment $e_0(v)$ and therefore also a lower disease environment at any given age, as compared to earlier born cohorts.

Thus, Eq. 1 can be restated as:

$$\frac{\mathrm{d}D_j(t,v)}{\mathrm{d}t} = \mu_j D_j(t,v) + \alpha_j e_0(v)\mathrm{e}^{-\beta t}. \tag{2}$$

For now, we consider only cohorts that were born during the epidemiological transition. Let $v(0) = v_0$ be the first cohort that experienced the epidemiological transition since birth and $e_0(v_0) = \epsilon$ the initial ETR at the start of the epidemiological transition such that

$$e_0(v) = \epsilon \mathrm{e}^{-\beta v}. \tag{3}$$

This expression captures in a simple way that later-born generations are exposed to lower ETR at any age and that this process diminishes as the epidemiological transition asymptotically comes to an end (for $v \to \infty$).

The differential equation (2) exhibits an explicit solution that provides health deficits of individuals as a function of age and the ETR at birth:

$$D_j(t,v) = D_{j0}\mathrm{e}^{\mu_j t} + \frac{\alpha_j e_0(v)\mathrm{e}^{\mu_j t}}{\mu_j + \beta}\left[1 - \mathrm{e}^{-(\mu_j+\beta)t}\right]. \tag{4}$$

The first term on the right-hand side of Eq. 4 reflects the familiar exponential increase of health deficits with age. Studies using the frailty index to measure health deficits usually estimate μ_j at about 3% per year. The term in front of the squared brackets shows that the effect of the initial level of the epidemiological transition ratio $e_0(v)$ on health deficits increases as the individual gets older. It is the gateway through which an individual's cohort (vintage) affects inflammaging and health deficit accumulation. At any given age t, later-born cohorts face a lower initial level of the epidemiological transition and thus a lower life-long exposure to infections. The last term in squared brackets captures that exposure to infectious diseases declines with age because the epidemiological transition advances as the individual gets older.

The main focus of the analysis is on the influence of the disease environment on death from non-communicable or chronic diseases. In the biology of aging death is explained by deteriorating health rather than age (Arking 2006) and it has been shown that there exists a strong log-log association between health deficits and the mortality rate from non-communicable diseases, the NCD mortality, denoted by $m_j^C(t,v)$, in which the superscript C stands for chronic diseases. This log-log association (or power law) can be expressed as:

$$m_j^C(t,v) = \eta_j D_j(t,v)^{\gamma_j}. \tag{5}$$

The health deficit elasticity of mortality γ_j is estimated with great precision at values around 3. An elasticity of 3 implies that the mortality rate increases at a rate of 9% per year when health deficits increase at a rate of 3% per year. This means that Eqs. 1 and 5 provide a biological foundation of Gompertz law, i.e., the (quasi-) exponential increase of the mortality rate with age (Gompertz 1825; Arking 2006; Hansen and Strulik 2025).

Following the related literature, we measure male excess mortality $\tilde{m}^C(t,v)$ by the male–female mortality ratio, only that we mostly focus on mortality from chronic (non-communicable) diseases:

$$\tilde{m}^C(t,v) \equiv \frac{m_M^C(t,v)}{m_F^C(t,v)}. \tag{6}$$

Additionally, we compute all-cause mortality $m_j(t,v)$, which, according to the model is simply the sum of the NCD mortality rate and the mortality rate from communicable (or infectious) diseases, $m_j(t,v) = m_j^C(t,v) + m_j^I(t,v)$. The infectious disease mortality rate is determined by the multiplicative interaction between the infectious disease environment $E(t)$ and the frailty of the individual measured by the health deficits (pre-conditions) of a person, $m_j^I(t,v) = E(t)\phi_j D_j(t,v)^{\delta_j}$.

Note that our model focuses on the continuous exposure to an infectious environment that gradually declines at a rate of β. In this respect, it differs fundamentally

from SIR models (Kermack and McKendrick 1927; Hethcote 2000). SIR models focus on acute infections and immunity from infections but neglect inflammaging, chronic health deficits, and human aging. They are an appropriate tool for studying short-run responses to pandemic shocks. In contrast, our model deliberately abstracts from such shocks and instead concentrates on the long-term effects of exposure to infections over the life course. This approach is more suitable for examining the impact of inflammaging on aging patterns of men and women and on male excess mortality from chronic diseases.

A stylized fact from several studies of human aging using the frailty index is that men develop new health deficits at a slightly faster rate, $\mu_M > \mu_F$. The health deficit elasticity of mortality, however, is slightly larger for women than for men, $\gamma_F > \gamma_M$ in Eq. 5 (see Mitnitski et al. 2002a, b; Dalgaard et al. 2022 for India see Krenz and Strulik 2023 and Tables A.1 and A.2 in the Supplementary Material). The influence of the epidemiological transition on male excess mortality of a cohort is obtained by the derivative $\mathrm{d}\tilde{m}^C(t,v)/\mathrm{d}e_0(v)$. Recalling that later-born generations face a lower initial ETR ratio $e_0(v)$, we conclude that male excess mortality increases in the course of the epidemiological transition if $\mathrm{d}\tilde{m}^C(t,v)/\mathrm{d}e_0(v) < 0$. We are now ready to state the main analytical result of the paper.

Proposition 1 *Male excess mortality from chronic diseases increases in the course of the epidemiological transition, that is* $\mathrm{d}\tilde{m}^C(t,v)/\mathrm{d}e_0(v) < 0$ *if*

$$\frac{\gamma_M}{\gamma_F} < \frac{\epsilon_{Fe}}{\epsilon_{Me}}, \tag{7}$$

in which ϵ_{je} *is the health deficit elasticity of sex* j *with respect to the initial state of the epidemiological transition,* $\epsilon_{je} \equiv \frac{\partial D_F(t,v)}{\partial e_0(v)} \frac{e_0}{D_F(t,v)}$, $j \in \{F, M\}$. *Under the (empirically plausible) assumptions* $\mu_M > \mu_F$ *and* $\gamma_F \geq \gamma_M$, *a sufficient condition for* Eq. 7 *is*

$$\frac{\alpha_F}{\alpha_M} > \frac{D_{F0}}{D_{M0}}. \tag{8}$$

Condition (7) is intuitive. It shows that male excess NCD mortality increases with declining infectious diseases if the female-male ratio of the elasticity of chronic health deficits with respect to infections ($\epsilon_{Fe}/\epsilon_{Me}$) is larger than the male–female ratio of the elasticity of mortality with respect to chronic health deficits (γ_M/γ_F). Condition (8) shows that this is the case if the female-male ratio of the inflamaging response to infections is sufficiently large. If initial health deficits for females do not exceed those of men, a sufficient condition is that $\alpha_F > \alpha_M$, meaning that females exhibit a stronger inflammaging response to infections than men. This means that the male–female NCD mortality ratio is highest without any infectious diseases, i.e., for $e_0 = 0$. With infections, the ratio is smaller because infections have a greater impact on female aging, driven by women's stronger inflammatory response. The inflammaging effect on mortality is greatest before the epidemiological transition, and it vanishes in the course of the transition, implying that the male–female ratio of NCD mortality increases.

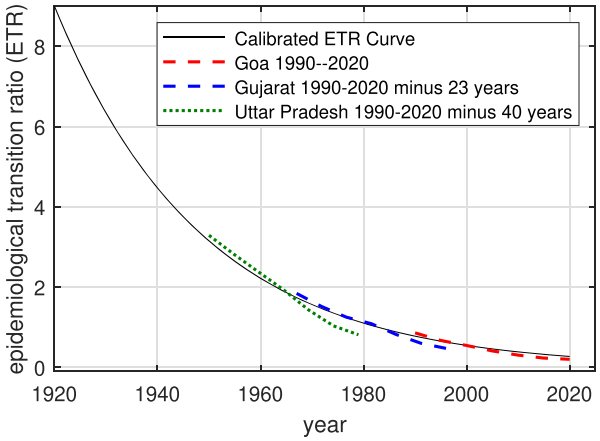

Fig. 4 Calibrated epidemiological transition. Black (solid) line: calibrated path of epidemiological transition level (ETR): 1920–2020. Data: Red (dashed) line: ETR Goa, 1990–2019; Blue (dash-dotted) line: ETR Gujarat, 1990–2019 minus 23 years; Green (dotted) line: ETR Uttar Pradesh, 1990–2019 minus 40 years. Data for both sexes from IHME (2021)

4 Male excess mortality during the Indian epidemiological transition: calibration and results

4.1 Calibration of the epidemiological transition

In order to compare model predictions and data, it is best to focus on an Indian state that has arrived at a low ETR at the end of the observation period. This ensures that sufficiently many generations have been exposed to an environment of declining infectious diseases. We pick Goa for our benchmark calibration. Goa has a low ETR and lies close to the regression line in Fig. 3, which means it is a state with about average response of excess male mortality to the epidemiological transition.

The GBD-I data provide observations of infectious disease mortality only back to 1990, meaning that the epidemiological transition was already well underway at the beginning of the observation period. The data compiled by Tumbe (2020) suggests that the epidemiological transition began around 1920, when infectious disease mortality began its sustained downward trend.[2] Tumbe (2020) shows that at that time deaths were to an overwhelming part caused by communicable diseases and that average Indian death rates started to decline from a level of about 45 per 1000, which is almost by a factor of 9 higher than the infectious disease death rate in 1990. We thus assume that the epidemiological transition started with an ETR of 9.0, which then declined by 3.5% per year such that the 1990–2019 part of the trajectory fits the observed path of Goa. In Fig. 4, the black line shows the calibrated ETR and the red dashed line shows the observed path of Goa.

[2] This estimate is consistent with Riley (2005) who also infers the 1920s as the onset of the health transition in India. Dyson (2018) also reports the first substantial declines of infectious disease mortality in India in the 1920s but anchors the start of the mortality transition in the 1940s. Notice, however, that our theory is independent to the specific choice for the onset of the transition.

We also fitted to the calibrated ETR curve the ETR paths of Gujarat (blue dashed line) and Uttar Pradesh (green dotted line). We see that the path that Gujarat followed in 1990–2019 fits approximately the calibrated path of Goa in 1967–1998, and that the path of Uttar Pradesh 1990–2019 fits the calibrated path for the 1950–1989 period. This means that we would match the paths of a slower follower state and an extreme latecomer state of the epidemiological transition in 1990–2019 by shifting the calibrated curve to the right by 23 and 40 years, respectively. The outward shifts would then measure the delay of the onset of the transition in these states compared to Goa.

4.2 Calibration of human aging and death

For the calibration of the biological parameters of the health deficit model and NCD mortality, we use results from Krenz and Strulik (2023), which are for convenience replicated as Tables A.1 and A.2 in the Supplementary Material. Using the same data as for the present paper, and recalling that age is measured in 5 year intervals, we see that the frailty index of Indian women (men) increased on average by 0.138/5=0.0276 (0.145/5=0.0290), when state and cohort fixed effects are taken into account (see columns 2 and 4 of Table A.1). We thus set $\mu_F = 0.0276$ and $\mu_M = 0.0290$. From the calibration of the epidemiological transition, we set $\beta = 0.035$.

In our benchmark exercises, we will consider men and women aged 45–49. This choice is motivated by the goal to have sufficiently many observations in the 1990–2019 period and to limit the potential bias of male excess mortality that could stem from maternal mortality. However, it should be noted that the focus on the 45–49 age group may not entirely eliminate all maternity-related effects on male excess mortality. On one hand, a selection effect may occur: women who experienced complications during childbirth and were in relatively poor health may have died earlier, leading to a healthier surviving female cohort and thus an increase of male excess mortality. On the other hand, a scarring effect could imply that women who survived childbirth complications may face increased mortality later in life, which would reduce male excess mortality.

For the calibration, we focus on the cohort aged 45–49 in 2010, which was born in 1965 (and the 4 years afterwards). At that time $e_0 = 1.8$, according to the calibration of the epidemiological transition (cf. Fig. 4). We take this value together with the values for β and the μ_js as specified above and calibrate the remaining parameters such that the predicted health deficits by age fit the data points for 2010. For the prediction, we use Eq. 4 starting at age 0. The model-individuals are thus exposed to infections and health deficit accumulation since birth, but we evaluate and compare predictions with data only for adults. This procedure leads to the estimates of $D_{M0} = D_{F0} = 0.090$, $\alpha_M = 1.22 \cdot 10^{-4}$ and $\alpha_F = 1.41 \cdot 10^{-4}$. Summarizing, we have that men age slightly faster than women, while women's aging is more affected by infectious diseases (α_F is 15% larger than α_M).

For the calibration of NCD death rates, we take Eq. 5 and the values of the mortality elasticities from Krenz and Strulik (2023), who estimated with high precision $\gamma_M = 2.90$ and $\gamma_F = 3.14$. The estimation results are replicated in Table A.2 in the Supplementary Material. A one percentage increase in health deficits is thus associated

with a slightly higher relative increase in NCD mortality of women. We calibrate the η_j's to match the data points from GBD-I for age-specific NCD mortality. This leads to the estimates $\log(\eta_M) = 9.35$ and $\log(\eta_F) = 9.58$. These values are close to the constants reported in Table A.2.

Our analysis focuses mainly on excess mortality for NCD deaths, but we also want to compare results for CD deaths and deaths from all causes. For that purpose, we refine (5) to calculate:

$$m_j^I(t, v) = e_0(v)e^{-\beta t} \cdot \phi_j D_j(t, v)^{\delta_j}, \tag{9}$$

CD Mortality thus depends on accumulated health deficits and the state of the epidemiological transition. We feed the calibrated values of β and $e_0(v)$ and the predicted age-paths of $D_j(t, v)$ from the benchmark calibration into the equation and set ϕ_j and δ_j to fit the actual CD death rates from the GBD-I data. This leads to the estimates $\delta_M = 3.8, \delta_F = 4.2, \log(\phi_M) = 11.91$, and $\log(\phi_F) = 12.57$, showing that, for given disease environment and given chronic health deficits, women die at a slightly higher rate from infectious diseases than men.

The predicted age profiles for health deficits, non-communicable disease mortality, and communicable disease mortality are shown by black solid lines in Fig. 5. Circles show the fitted data points. Red dashed lines show predictions when e_0 is by 50% higher, and blue dash-dotted lines show predictions for 50% lower e_0. For the calibrated value of $\beta = 0.035$ this means that individuals reflected by the red line (blue line) are born 20 years earlier (20 years later) than the benchmark cohort. Later-born individuals face, at any given age, a lower ETR, and they therefore "naturally" die at lower rates from infections. More interestingly, the model also predicts that — due to the lower lifetime exposure to infections — later-born individuals also die at lower rates from non-communicable diseases (center panels in Fig. 5). Figure A.6 in the Supplementary Material replicates the results for logarithmic scaling of deficits and mortality rates.

4.3 The rise of excess male mortality

We are now ready to present and discuss the results. We first feed into the model a series of alternative $e_0(v)$-values from the interval 3.7 to 1.3. Given the calibrated course of the epidemiological transition, this means that we focus on cohorts born between 1945 and 1965 who celebrate their 45th birthday between 1990 and 2020. For these cohorts, we computed the predicted male and female NCD mortality rate and the implied male excess mortality at age 45 from 1990 to 2020, which we then compared with the GBD-I data.

The results are shown in Fig. 6 in the panel on the left-hand side. The blue solid line shows the model's prediction. The circles show the excess mortality computed for Goa from the GBD-I data, and the red dashed line shows the linear fit for these data. The model's prediction captures the trend of excess NCD mortality fairly accurately, though it slightly overestimates the overall level.

Notice that our controlled computational experiment varies only the level of the epidemiological transition at the birth of a cohort and shuts down any other potential

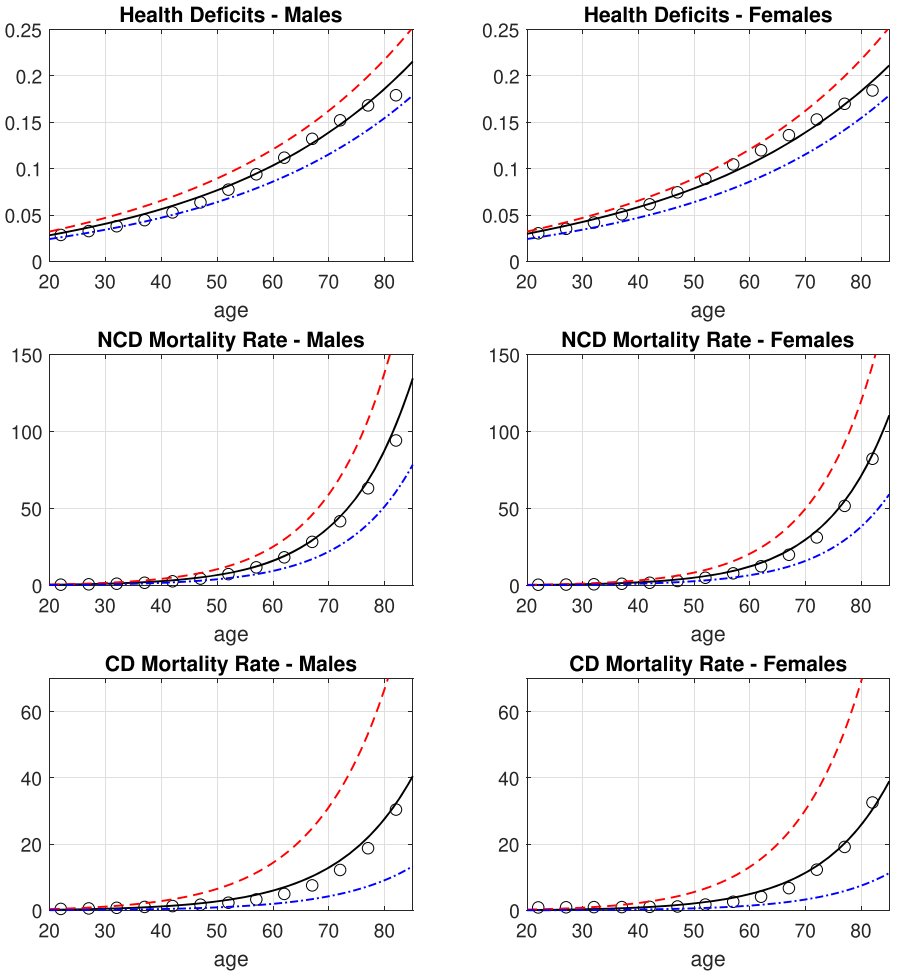

Fig. 5 Calibrated aging and death of men and women. Predicted health deficit accumulation, NCD mortality, and CD mortality for men and women for alternative paths of the epidemiological transition ratio (ETR), parameterized as $ETR = e_0 \exp(-0.035 \cdot t)$, where t is time in years and with $e_0 = 1.8$ (black solid lines), $e_0 = 2.4$ (red dashed lines), and $e_0 = 1.2$ (blue dotted lines). Circles: data averages computed from IHME (2021). Health deficits are measured by the frailty index. Age-group specific mortality rates are measured as deaths per 1,000

influence. We can thus conclude that the model is capable to fully explain the increase of excess mortality due to the decline in infectious diseases and their impact on biological aging (accumulation of chronic health deficits) of men and women. The reason is that women have an NCD mortality advantage over men due to their slower biological aging in an infection-free environment ($\mu_F < \mu_M$) while they are subject to greater inflammaging responses to infections during the epidemiological transition ($\alpha_F > \alpha_M$). Women, therefore, benefit more than men from the decline in infectious diseases in terms of fewer chronic health deficits and fewer deaths from NCDs.

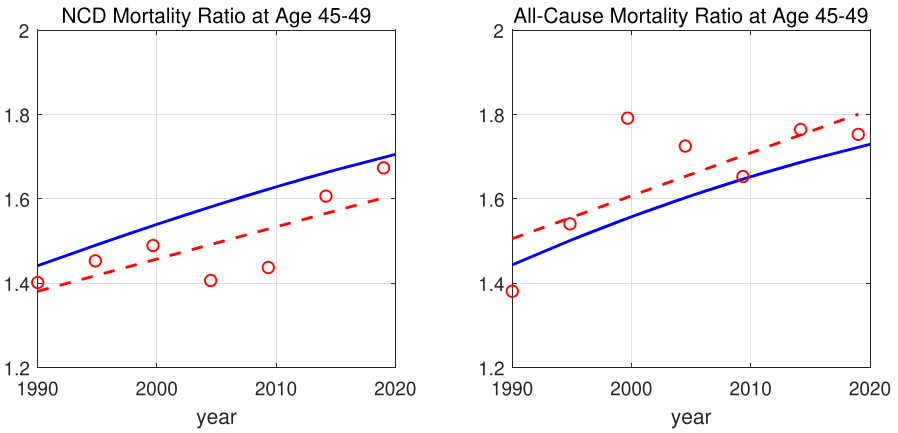

Fig. 6 Male–female mortality ratio at age 45–49 by year: data vs. prediction. Prediction (blue solid lines): calibrated model with an initial ETR of 3.7 in 1945 (when the generation aged 45 in 1990 was born) and a new generation born every 5 years until 1975. Circles: Data for Goa and both sexes from IHME (2021). Red dashed lines: fitted regression lines. NCD Mortality is mortality from non-communicable diseases

The panel on the right-hand side of Fig. 6 shows the predicted excess mortality of men with respect to death from all causes (blue solid line). All causes in our model are the sum of the death rates from communicable and non-communicable diseases. The data also includes injuries, but these only account for a minor proportion of deaths. Data points are shown by circles, and the red dashed line shows the estimated trend. Once again, the model captures the general trend reasonably well, though it slightly underestimates the level of excess mortality.

We next examine the model's prediction with respect to age and cohort effects. We first consider again the cohort born in 1945 (with the calibrated $e_0(v) = 3.7$). Solid black lines in Fig. 7 show for all adult ages the predicted excess mortality for NCD deaths and deaths from all causes. The line is mildly negatively sloped in alignment with the estimates from Table 1. To understand the negative slope, notice that with advancing age also the epidemiological transition proceeds such that the self-productivity of health deficits becomes the dominating effect in old age. This means that men accumulate health deficits faster than women (since $\mu_M > \mu_F$), however, the health deficit elasticity of mortality is larger for women, which is the dominating effect. To see this formally, we shut down the epidemiological transition by setting $e^{-\beta t} = 0$ (which is asymptotically true for $t \to \infty$). Then, after inserting (2) and (5) into excess mortality we have $\tilde{m}(t) = e^{(\mu_M\gamma_M - \mu_F\gamma_F)t}$, which declines in t since $\mu_M\gamma_M < \mu_F\gamma_F$.

We next consider a cohort born 20 years later, in 1965, and thus subject to a lower initial ETR of 1.8. As shown by the dashed black lines, the predicted excess mortality of men is higher at all ages. The reason is that the later-born cohort is exposed to fewer infectious diseases at any given age and thus the inflammaging effects are smaller, which disproportionally improves the survival of women. Finally, red lines show the trend lines obtained from regressions for Goa with the GBD-I data (14 observations, $R^2 = 0.62$ for NCD mortality and $R^2 = 0.54$ for all causes). In these trend lines,

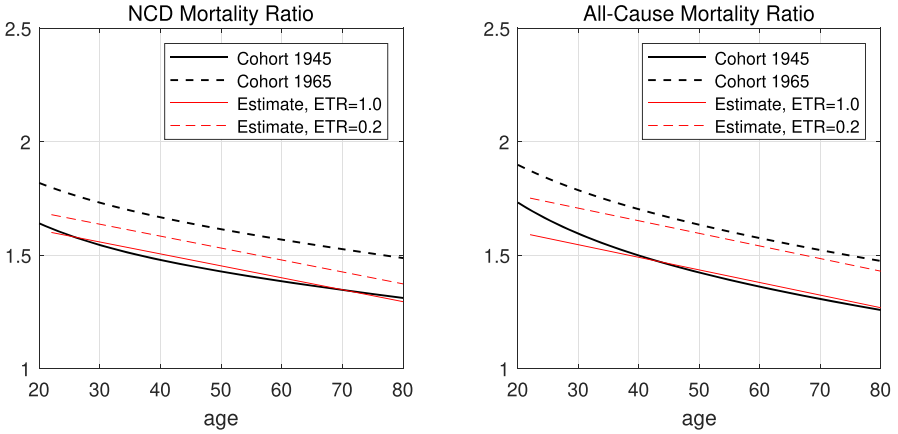

Fig. 7 Male–female mortality ratio by age and cohort: data vs. prediction. Prediction: black solid lines: initial ETR 3.7 in 1945 (when the generation aged 45 in 1990 was born); black dashed lines: initial ETR 1.8 in 1965 (when the generation age 45 in 2010 was born). Red lines: regression results from GBD-I data. NCD Mortality is mortality from non-communicable diseases

the ETR ratio is a shifter. The solid red lines show the regression result for ETR$=1$ (about the average level experienced by the 1945 cohort). The model prediction and the data are well matched in terms of the level and slope of the curve. The dashed red line shows the regression result for ETR$=0.2$, which could be an average ETR experienced by the 1965 cohort. The model overpredicts the effect on excess mortality with regard to NCD deaths, but it is well-aligned with the estimated effect on excess mortality from all causes.

4.4 Excess mortality across states and in history

Finally, we consider two "out-of-sample" predictions. In the first computational experiment, we feed into the model 30 epidemiological transitions that proceed at the same speed ($\beta = 0.035$) but started in 1970 from different initial ETRs, linearly spaced in the interval between 0.9 and 5.5. These values can be thought of as the initial ETRs in the 30 states in 1970, which is the birth year of the cohort that was 45 years old in 2015. We then collect the predicted excess mortality of men at age 45, subtract the mean across states, and plot it against the predicted state-specific ETR in 2015. The result is shown by blue circles in Fig. 8. The black dashed line shows the result of a linear regression of excess mortality on ETR across states, with an estimated slope of 0.45.

The results can be compared with the regression results for India from Fig. 3. Naturally, the model predictions are closer to the regression line because, in the model, the ETR level is the only difference across states. The model predicts about correctly the range of excess mortality across states and the negative association of ETR and excess mortality across states. However, it overpredicts the strength of the state-ETR effect on excess mortality.

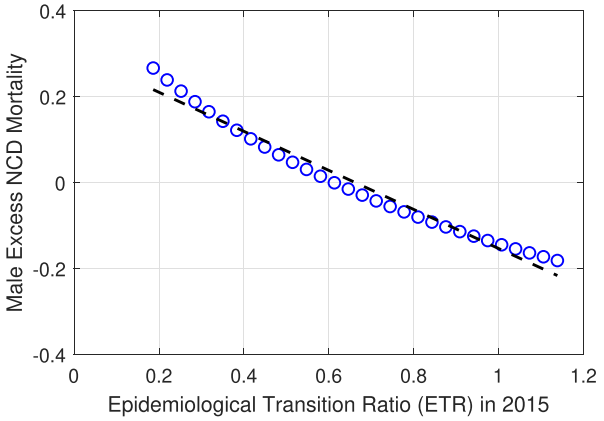

Fig. 8 Predicted excess NCD mortality by state. The figure shows the results of a simulation for 30 different initial values of the epidemiological transition in 1970. It plots the implied ETR value in 2015 and the implied male excess mortality at age 45 in its deviation from the mean. The dashed line shows the implied linear regression line: $0.30 - 0.45ETR$

In our final computational experiment, we simulate excess mortality of men for Indian cohorts born from 1870 to 1970. In this bold exercise of comparative dynamics, we keep everything as calibrated for the Goa 1945 generation and the calibrated epidemiological transition from Fig. 4. Since the calibrated epidemiological transition begins in 1920, the cohorts born before 1920 lived a part of their life in a stationary world. For example, the cohort from 1870 faced for 50 years the high pre-transition ETR and (the survivors) experienced the transition only in old age. The cohorts born 1870–1875 did not experience any decline of infections before their 45th birthday (and they thus look identical to the omitted cohorts born before 1870). Cohorts born from 1920 onwards faced a continuous epidemiological transition from birth, and later cohorts faced a less infectious environment at any given age.

Results are shown in Fig. 9. The panel on the left-hand side shows the log of mortality from communicable diseases for men and women at age 45. Female mortality

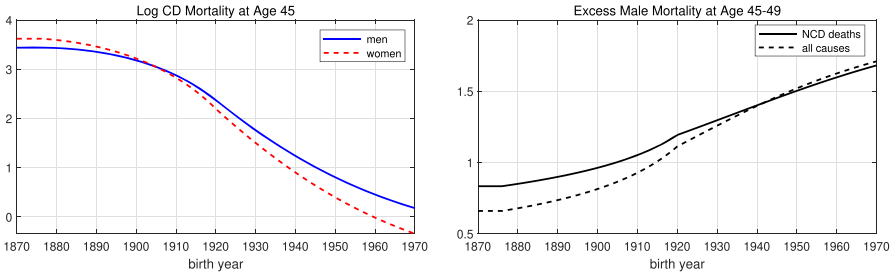

Fig. 9 Predicted male and female CD mortality and male excess mortality by birth year. Left: logarithm of mortality at age 45 from non-communicable diseases for men (blue solid line) and women (red dashed line). Right: male/female mortality at 45 for non-communicable diseases and all causes. Epidemiological Transition as calibrated in Fig. 4

lies initially above that of men but declines more quickly with increasing exposure to the epidemiological transition. The panel on the right-hand side shows the predicted excess mortality of men for NCD deaths (solid line) and for deaths from all causes (dashed line). The model predicts a female disadvantage in survival for the early-born cohorts, in line with the observation of cohort effects in Fig. 2. Male mortality for deaths from all causes breaks even for the cohort born in 1915, about 35 years later than it has been observed by Beltran-Sanchez et al. (2015) for a sample of developed countries. More importantly, the model predicts that the decline in infectious diseases led to a steady decline of non-communicable diseases long before the cardiovascular revolution of the 1970s (as shown for the US by Costa 2000). Because women benefited more from reductions in inflammaging, excess mortality from NCDs increased in step with excess mortality from all causes.

5 Discussion and conclusion

In this paper, we examined for the first time the association between an ongoing epidemiological transition and excess mortality of men in a developing country. Using panel data from IHME (2021) for India, we showed how excess mortality increased over time and with the birth year of cohorts born 1900–1970. We showed how these developments are associated with the epidemiological transition such that, for a given age, excess mortality is higher in later-born cohorts, i.e., for cohorts that faced a less infectious disease environment over their lifetime. We then developed a theory that explains these observations by integrating an inflammaging channel into the health deficit model of human aging and mortality. We showed under which conditions a greater inflammation response of women is a sufficient condition for emerging male excess mortality in the course of the epidemiological transition. We then calibrated the model with the GBD-I data and showed that the epidemiological transition can account for the increase in male excess mortality and its association with the birth year and age of individuals, as well as with the level of the epidemiological transition across the Indian states. In conclusion, our study established the inflammaging channel as a powerful explanation for the increasing excess mortality of men.

A limitation of our study is that the structure of the GBD-I data does not permit analysis of within-state variation in physiological aging, mortality, or the progression of the epidemiological transition. Additionally, the disability weights used in the calculation of DALYs are fixed across age groups and time periods, and are based on international survey data without India-specific adjustments. While these constraints introduce measurement error that affects the estimates of total DALYs — and thereby influences the prioritization of health conditions for policy — such biases are less likely to affect the dynamics of state-specific ETRs, which are central to our analysis. Furthermore, we demonstrated that our main findings remain robust when the ETR is substituted with male or female mortality from communicable diseases as an alternative indicator of the epidemiological transition.

Like other research based on GBD-I data, our analysis is also constrained by uncertainties in location- and disease-specific estimates (Dandona et al. 2017). The Civil Registration System (CRS), a major input for the GBD, remains incomplete in many

Indian states, with death registration rates falling below 50% in some regions as recently as 2015. Underreporting tends to be more severe among marginalized populations, including women, which can introduce bias in the estimation of male excess mortality. While the GBD uses sophisticated modeling techniques to adjust for missing data, the underlying inputs are not always transparent, and the models themselves rely on assumptions that may not fully capture India's regional and demographic heterogeneity. For instance, cause-of-death data for large northern states like Uttar Pradesh and Bihar are not based on comprehensive medical certification, but rather on verbal autopsy data from the Sample Registration System, extrapolated using statistical models that may not capture regional epidemiological profiles accurately. These models often borrow strength from better-documented regions, such as Kerala or Tamil Nadu, which can lead to systematic misestimation of disease burdens in poorly covered states. This limits the quantitative precision of our subnational calculations of male excess mortality and suggests that findings related to state-level trends should be interpreted with caution. Nonetheless, these data constraints do not undermine the broader conclusion that the epidemiological transition has contributed significantly to the rise in male mortality.

Every successful application of a theory involves Occam's razor: it highlights one causal pathway of a phenomenon under the assumption of no other mechanisms. The most important question is then whether this assumption is crucial for the results. In our case, the literature has emphasized that Indian males frequently receive better nutrition and health care than females. This means that the male–female mortality ratio increases if discrimination against girls and women decreases with economic development. However, this channel is likely to complement and even accelerate the inflammaging channel since better nutrition and health care protect against infections.

The nutrition-infection channel could be explored further in future research by utilizing the model of childhood development by Dalgaard et al. (2021). Building on Barker's (1995) hypothesis, Hansen et al. link ontogenetic growth during the prenatal and childhood periods with the accumulation of health deficits in old age and offer a unified theory of how early-life shocks affect late-life health and longevity. In its current version, the model emphasizes nutrition and does neither address infections nor gender differences but it could be extended to include the inflammaging mechanism and interaction effects between disease environments and physiological development (Dewey and Mayers 2011; Morales et al. 2023). In this model, the provision of childhood nutrition is explained by parental preferences, which may be biased against girls. Reducing gender bias would improve nutritional outcomes for females, enhancing their resistance to disease in both childhood and adulthood, and thereby potentially amplifying the observed increase in male excess mortality.

Since the 1970s, the epidemiological transition in India has been accompanied by the fertility transition, which provides an alternative and complementary pathway of increasing male excess mortality. Maternal mortality is strongly positively associated with fertility (Conde-Agudelo et al. 2006; Cleland et al. 2012; WHO 2019). In high-fertility settings, elevated maternal mortality effectively masked women's biological longevity advantage. As fertility declined and maternal health improved, these external mortality risks diminished, allowing underlying biological differences in survival to become more apparent. Consistent with this mechanism, historical data show that male

and female mortality rates at reproductive ages were approximately equal prior to the fertility transition, but diverged thereafter (Wisser and Vaupel 2014; Charbonneau 1993). Notice, however, that — by definition — maternal mortality is not classified as non-communicable disease (NCD) mortality and that our study focuses on explaining the rising excess NCD mortality of men.

As noted in the Introduction, a limitation of the current model is its omission of gender differences in health behaviors. Research on male excess mortality in developed countries has shown that part of the gender mortality gap can be attributed to earlier or greater consumption of unhealthy goods by men (Beltran-Sanchez et al. 2015). Another promising avenue for future work is thus to integrate the inflammaging pathway into a model of gender-specific health behavior, for instance, using the framework developed by Schünemann et al. (2017). That study extended the health deficit model to account for the role of gender differences in physiology, preferences, and income in explaining male excess mortality in the US. Through counterfactual simulations (by assigning women male-like preferences), the study found that differences in health behaviors could explain up to 80% of the gender gap in life expectancy. The study neglected infectious diseases and inflammaging, which was recently explored by Strulik and Grossmann (2024) who also considered gender differences in protection from infections (e.g., through vaccination) but neglected unhealthy behavior. Most importantly, both studies focused on a high-income country, where the epidemiological transition is largely complete.

The model of Schünemann et al. (2017) also offered an explanation for why the gender gap tends to narrow again in rich countries: women are characterized by higher relative risk aversion — reflected in more concave utility functions — which leads to a stronger emphasis on health and longevity and less inclination toward short-term gratifications such as smoking and alcohol consumption. However, as income rises, this effect diminishes because the slopes of the utility functions of men and women converge, becoming similarly flat. Applied to India, these findings suggest that part of the current excess mortality may result from gender differences in health behavior. They also indicate that male excess mortality is likely to increase further even if the inflammaging effect fades once the epidemiological transition is complete. Eventually, however, it will likely decline again, following the pattern observed in the richest countries.

Supplementary Information The online version contains supplementary material available at https://doi.org/10.1007/s00148-025-01124-0.

Acknowledgements We would like to thank Nicole Bittermann for excellent research assistance and Martin Anthony, Jesus Crespo Cuaresma, Qi Cui, Matthias Doepke, Martin Karlsson, Kaveh S. Nobari, Johannes Ruf, Eric Schneider, Wen Su, Sebastian Vollmer, four anonymous reviewers, and the editor, Oded Galor, for discussion and comments.

Funding Open Access funding enabled and organized by Projekt DEAL.

Declarations

Competing interests The authors declare no competing interests.

References

Abeliansky A, Strulik H (2018) How we fall apart: similarities of human aging in 10 European countries. Demography 55:341–359

Abeliansky A, Erel D, Strulik H (2020) Aging in the USA: similarities and disparities across time and space. Sci Rep 10(1):14309

Arking R (2006) The biology of aging: observations and principles. Oxford University Press, Oxford

Austad SN (2006) Why women live longer than men: sex differences in longevity. Gend Med 3(2):79–92

Barker DJ (1995) Fetal origins of coronary heart disease. BMJ 311(6998):171–174

Bektas A, Schurman SH, Sen R, Ferrucci L (2018) Aging, inflammation and the environment. Exp Gerontol 105:10–18

Beltran-Sanchez H, Finch CE, Crimmins EM (2015) Twentieth century surge of excess adult male mortality. Proc Natl Acad Sci 112(29):8993–8998

Blodgett JM, Theou O, Howlett SE, Wu FC, Rockwood K (2016) A frailty index based on laboratory deficits in community-dwelling men predicted their risk of adverse health outcomes. Age Ageing 45(4):463–468

Bloom DE, Cafiero-Fonseca ET, McGovern ME, Prettner K, Stanciole A, Weiss J, Bakkila S, Rosenberg L (2014) The macroeconomic impact of non-communicable diseases in China and India: Estimates, projections, and comparisons. J Econ Ageing 4:100–111

Bloom DE, Sekher TV, Lee J (2021) Longitudinal Aging Study in India (LASI): new data resources for addressing aging in India. Nat Aging 1(12):1070–1072

Canudas-Romo V, Saikia N, Diamond-Smith N (2015) The contribution of age-specific mortality towards male and female life expectancy differentials in India and selected States, 1970-2013. Asia-Pac Popul J 30(2)

Case A, Paxson C (2005) Sex differences in morbidity and mortality. Demography 42(2):189–214

Charbonneau H (1993) The first French Canadians: pioneers in the St. University of Delaware Press, Lawrence Valley

Chatterji S, Kowal P, Mathers C, Naidoo N, Verdes E, Smith JP, Suzman R (2008) The health of aging populations in China and India. Health Aff 27(4):1052–1063

Chaudhary M, Arokiasamy P (2019) Patterns of frailty and quality of life among older adults: Comparative analysis using SAGE States of India. J Popul Ageing 12(1):1–23

Cleland J, Conde-Agudelo A, Peterson H, Ross J, Tsui A (2012) Contraception and health. Lancet 380(9837):149–156

Conde-Agudelo A, Rosas-Bermudez A, Kafury-Goeta AC (2006) Birth spacing and risk of adverse perinatal outcomes: a meta-analysis. JAMA 295(15):1809–1823

Costa DL (2000) Understanding the twentieth-century decline in chronic conditions among older men. Demography 37(1):53–72

Costa DL (2002) Changing chronic disease rates and long term declines in functional limitation among older men. Demography 39(1):119–137

Crimmins EM, Shim H, Zhang YS, Kim JK (2019) Differences between men and women in mortality and the health dimensions of the morbidity process. Clin Chem 65(1):135–145

Dalgaard C-J, Strulik H (2014) Optimal aging and death: understanding the Preston curve. J Eur Econ Assoc 12:672–701

Dalgaard CJ, Hansen CW, Strulik H (2021) Fetal origins - a life cycle model of health and aging from conception to death. Health Econ 30(6):1276–1290

Dalgaard C-J, Hansen C, Strulik H (2022) Physiological aging around the world. PLoS ONE 17(6):e0268276

Dandona L, Dandona R, Kumar GA, Shukla DK, Paul VK, Balakrishnan K et al (2017) Nations within a nation: variations in epidemiological transition across the states of India, 1990–2016 in the Global Burden of Disease Study. Lancet 390(10111):2437–2460
Dewey KG, Mayers DR (2011) Early child growth: how do nutrition and infection interact? Matern Child Nutr 7:129–142
Dyson T (2018) A population history of India: from the first modern people to the present day. Oxford University Press
Ferrucci L, Fabbri E (2018) Inflammageing: chronic inflammation in ageing, cardiovascular disease, and frailty. Nat Rev Cardiol 15(9):505–522
Finch CE, Crimmins EM (2004) Inflammatory exposure and historical changes in human life-spans. Science 305(5691):1736–1739
Finch CE (2010) Evolution of the human lifespan and diseases of aging: roles of infection, inflammation, and nutrition. Proc Natl Acad Sci 107(suppl 1):1718–1724
Franceschi C, Campisi J (2014) Chronic inflammation (inflammaging) and its potential contribution to age-associated diseases. J Gerontol Ser A Biomed Sci Med Sci 69(Suppl 1):S4–S9
Franceschi C, Garagnani P, Morsiani C, Conte M, Santoro A, Grignolio A, Monti D, Capri M, Salvioli S (2018) The continuum of aging and age-related diseases: common mechanisms but different rates. Front Med 5:1–23
GBD-I (2017) Indian Council of Medical Research, Public Health Foundation of India, and In stitute for Health Metrics and Evaluation. India: Health of the Nation's States - The India State-level Disease Burden Initiative. New Delhi, India: ICMR, PHFI, and IHME; 2017. https://vizhub.healthdata.org/gbd-compare/india
Goldin C, Lleras-Muney A (2019) XX XY?: the changing female advantage in life expectancy. J Health Econ 67:102224
Gompertz B (1825) On the nature of the function expressive of the law of human mortality, and on a new mode of determining the value of life contingencies. Philos Trans R Soc Lond 115:513–583
Hansen CW, Strulik H (2025) How do we age? A decomposition of Gompertz law. J Health Econ 101:102988
Hethcote HW (2000) The mathematics of infectious diseases. SIAM Rev 42(4):599–653
HLD (2025) Human life-table database. https://www.lifetable.de/Country/Country?cntr=IND#:~:text=1901%20
Holden C (1987) Why do women live longer than men? Science 238(4824):158–160
Hosseini R, Kopecky KA, Zhao K (2022) The evolution of health over the life cycle. Rev Econ Dyn 45:237–263
Howlett SE, Rutenberg AD, Rockwood K (2021) The degree of frailty as a translational measure of health in aging. Nat Aging 1(8):651–665
IHME (2021) Institute for health metrics and evaluation (IHME). GBD compare data visualization. Global Burden of Disease (GBD) Study 2021. Available from http://vizhub.healthdata.org/gbd-compare. (Accessed 22 Jan 2022)
Kermack WO, McKendrick AG (1927) A contribution to the mathematical theory of epidemics. Proc R Soc Lond A 115(772):700–721
Klein SL, Flanagan KL (2016) Sex differences in immune responses. Nat Rev Immunol 16(10):626–638
Krenz A, Strulik H (2023) Physiological aging in India: the role of the epidemiological transition. PLoS ONE 18(7):e0287259
Mitnitski AB, Rockwood K (2016) The rate of aging: the rate of deficit accumulation does not change over the adult life span. Biogerontology 17(1):199–204
Mitnitski AB, Mogilner AJ, Rockwood K (2001) Accumulation of deficits as a proxy measure of aging. Sci World 1:323–336
Mitnitski AB, Graham JE, Mogilner AJ, Rockwood K (2002) Frailty, fitness and late-life mortality in relation to chronological and biological age. BMC Geriatr 2(1):1–8
Mitnitski AB, Mogilner AJ, MacKnight C, Rockwood K (2002) The accumulation of deficits with age and possible invariants of aging. Sci World J 2:1816–1822
Morales F, Montserrat-de la Paz S, Leon MJ, Rivero-Pino F (2023) Effects of malnutrition on the immune system and infection and the role of nutritional strategies regarding improvements in children's health status: a literature review. Nutrients 16(1):1
Oksuzyan A, Juel K, Vaupel JW, Christensen K (2008) Men: good health and high mortality. Sex differences in health and aging. Aging Clin Exp Res 20(2):91–102

Patel R, Srivastava S, Kumar P, Chauhan S, Govindu MD, Jean Simon D (2021) Socio-economic inequality in functional disability and impairments with focus on instrumental activity of daily living: a study on older adults in India. BMC Public Health 21(1):1–13
Rieker PP, Bird CE (2005) Rethinking gender differences in health: why we need to integrate social and biological perspectives. J Gerontol Ser B 60:40–47
Riley JC (2005) The timing and pace of health transitions around the world. Popul Dev Rev 31(4):741–764
Rockwood K, Mitnitski A (2007) Frailty in relation to the accumulation of deficits. J Gerontol A Biol Sci Med Sci 62(7):722–727
Santoro A, Bientinesi E, Monti D (2021) Immunosenescence and inflammaging in the aging process: age-related diseases or longevity? Ageing Res Rev 71:101422. https://doi.org/10.1016/j.arr.2021.101422
Schünemann J, Strulik H, Trimborn T (2017) The gender gap in mortality: how much is explained by behavior? J Health Econ 54:79–90
Searle SD, Mitnitski A, Gahbauer EA, Gill TM, Rockwood K (2008) A standard procedure for creating a frailty index. BMC Geriatr 8(1):24
Strulik H, Grossmann V (2024) The economics of aging with infectious and chronic diseases. Econ Hum Biol 101319
Tumbe C (2020) Pandemics and historical mortality in India (No. WP 2020-12-03). Indian Institute of Management Ahmedabad, Research and Publication Department
UN (2022) United nations population division. World Population Prospects 2022. https://population.un.org/wpp/Download/Standard/Population/
UN (2024) United nations population division. World Population Prospects 2024
Verbrugge LM (1985) Gender and health: an update on hypotheses and evidence. J Health Soc Behav 26:156–182
Waldron I (1993) Recent trends in sex mortality ratios for adults in developed countries. Soc Sci Med 36(4):451–462
Wardle J, Haase AM, Steptoe A, Nillapun M, Jonwutiwes K, Bellisie F (2004) Gender differences in food choice: the contribution of health beliefs and dieting. Ann Behav Med 27(2):107–116
Wisser O, Vaupel JW (2014) The sex differential in mortality: a historical comparison of the adult-age pattern of the ratio and the difference. Max Planck Institute for Demographic Research Working Paper
WHO (2019) Trends in maternal mortality 2000 to 2017: estimates by WHO, UNICEF, UNFPA. World Bank Group and the United Nations Population Division, World Health Organization, Geneva
Yang Y, Kozloski M (2011) Sex differences in age trajectories of physiological dysregulation: inflammation, metabolic syndrome, and allostatic load. J Gerontol Ser A Biomed Sci Med Sci 66(5):493–500