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OPEN The role of age inequalities in cause of death in the slow pace of epidemiological transition in India

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In developed countries, low disparity in lifespan contributed by the reduction in the burden of noncommunicable diseases (NCDs) is the key to advances in epidemiological transition. Contrarily, India passing through a phase of the dual burden of CDs and NCDs shows a heavy burden of NCDs responsible for the high disparity in lifespan. The Gini coefficient was decomposed for examining the contribution of 22 causes of death and their repercussions for inequality in age at death for 30 years between 1990–1994 and 2015–2019, using Global Burden of Disease data. The outcomes of the study reveal that India's epidemiological transition has been just modest on account of high inequality in mortality by NCDs emplaced in the middle through old age despite a consistent mortality decline at infant through old age for communicable diseases (CDs). The structural changes in causes of death structure is shaped by CDs rather than NCDs, but overall bolstered by the adult mortality decline, especially in women. However, the process is restrained by the small contribution of the middle age group and a benign contribution of old mortality decline owing to the low threshold age. India needs to target health interventions in seeking significant mortality decline in the middle age group of 50–69 years that is warranted for epidemiological transition apace as evident in the developed nations.

Abbreviations

GBD	Global burden of diseases
IHME	Institute for health metrics and evaluation
NCDs	Noncommunicable diseases
CDs	Communicable diseases
ASDR	Age-specific death rates
ACSDR	Age-cause-specific death rates
ICD	International classification of diseases
COPD	Chronic obstructive pulmonary disease
IMR	Infant mortality rate
U5MR	Under-five mortality rate
MMR	Maternal mortality ratio
MoHFW	Ministry of health and family welfare
NPCDCS	National programme for prevention and control of cancer, diabetes, cardiovascular diseases and
	strokes
MCCD	Medical certification of causes of death
SRS	Sample registration system
HMIS	Health management information system
NPHCE	National programme for health care of elderly
NMAP	National multisectoral action plan

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22 causes of death

CRD	Chronic respiratory diseases
CVDs	Cardiovascular diseases
Dia&k	Diabetes and kidney diseases
DigeD	Digestive diseases
Enter	Enteric (diarrhea and typhoid) infections
HIV/A	HIV/AIDS and STI
Mat	Maternal disorders
Menta	Mental disorders
Muscu	Musculoskeletal disorders
Neo	Neonatal disorders
Neopl	Neoplasms
Neuro	Neurological disorders
NTD&M	Neg. tropical diseases and malaria
NutrD	Nutritional deficiencies
ONCDs	Other noncommunicable diseases
Otinf	Other infectious diseases
RI&T	Respiratory infections and tuberculosis
SelHV	Self-harm and interpersonal violence
SkiSu	Skin and subcutaneous diseases
SubUs	Substance use disorders
Trans	Transport injuries
Unint	Unintentional injuries

Many developed countries in the past long-term have conformed with the course of epidemiological transition as described by Omran's theory^{1,2}. Omran's epidemiological transition² ardently advocates the substitution of communicable diseases (CDs) by the heavy burden of noncommunicable diseases (NCDs) while passing from the phase of 'Age of Receding Pandemics', and until the 'Age of Degenerative and Man-made Diseases'. However, aberrations are also highlighted³⁻⁷, especially with the increase in the pathogenesis and aetiology of chronic diseases and also, imbricated causations across infectious and chronic diseases⁸⁻¹⁰ that are entwined with man-made diseases¹¹. The epidemiological transition shows strong linkages with the advances in mortality transition intrigued by the causes of death structure^{12,13}. Structural changes in causes of death and progression in epidemiological transition is majorly driven by the larger reduction in premature deaths at ages^{14,15} below the threshold age that separates premature deaths from old ages¹⁶. The same diseases show affirmative (positive) role at premature ages whereas show opposing (negative) roles in old ages^{17,18} owing to the threshold age¹⁹. This disparateness of affirmative and dissenting roles about the threshold age of same causes of death is conspicuous in the analysis of lifespan disparity (e^{\dagger}) or inequality in age at death^{20,21} measured by Gini coefficient at birth $(G_0)^{22,23}$ as compared to life expectancy at birth (e₀). Developed countries passing through low mortality regime have shown significant developments in the causes of death structure, low inequality in age at death²⁴, and mortality and/or morbidity compression^{25,26}; however, these phenomena stand modest in developing countries²⁷. Among these fundamental phenomena, exploration of inequality in age at death by age-sex and causes of death deciphers scrupulous assessments of the structural changes in causes of death and hence the progress of epidemiological transition.

Developed countries such as the European Unions, New Zealand, Japan, and England & Wales experience apace in epidemiological transition because of greater reduction in premature mortality, high threshold age, and swift structural changes in cause of death²⁸. The USA's health policies asserted for the reduction in burden of chronic NCDs²⁹; however, the results over time exhibited higher e^{\dagger} and lower e_0 compared to other developed countries^{30,31}. Shkolnikov et al.²³ demonstrate higher G_0 values in the USA than in the UK attributable to lesser reduction in premature mortality though both countries show similar threshold age, i.e. 80 years and above³². However, developing countries such as India and South Africa show the threshold age of 71.3 years and 58.7 years, respectively³². A low threshold age provides a narrow age-interval for reduction in premature mortality. So, a lag of ten and more years in the threshold age restraints the affirmative contribution of premature ages and moreover, augments negative repercussions of middle and old ages on mortality reductions in developing countries. As a consequence, a reduction in the premature mortality is subdued and the structural changes in causes of death is constrained. Over and above that, developing country India has the challenge of economic cost for the causes of death surveillance³³, along with the cognisance of health transition³⁴ and morbidity expansion³⁵.

Smits and Monden³⁶ demonstrates that the diffusion hypothesis is more conclusive while it is contingent upon the reduction in premature mortality. The oversimplification of the Omran's model is not justified in the cognisance of the welfare Kuznets curve³⁷. A developing country India do achieve on the higher survival of infants, children, and mothers³⁸ to reducing premature mortality through long-term health programmes and policies³⁹. However, NCDs intruded in the mid-1990s, and since thereupon premature mortality becomes a big concern in adult through old ages leading to dual burden of diseases in India⁴⁰. Inevitably, the country attests a languid progression in mortality compression²⁷ enduring large premature mortality and slow mortality deceleration^{41,42}. Therefore, it is crucial to examine the role of the heavy burden of chronic NCDs^{43,44} and also injuries⁴⁵ over a wide age range in adult through old ages for the progression in epidemiological transition in India.

The study assesses the role of inequality in age at death by causes of death for advances in epidemiological transition in India during a period of 30 years between 1990–1994 and 2015–2019. We tested the hypotheses that (a) whether chronic NCDs versus communicable diseases (CDs) contributes to higher inequality in age at death, (b) whether mortality decline in adult and higher age groups is crucial for structural changes in causes of

death in high versus low mortality regimes; and, together they construe the prolonged dual burden of disease in India. The specific objectives of the study are (1) to assess the age- and cause-specific contribution to the changes in life expectancy at birth (Δe_0) and inequality in age at death (ΔG_0) and (2) to examine the transformation in age at death by age, sex, and causes of death. The study aims to explore the phenomenal phase of dual burden of disease by inequalities in mortality by causes of death and hence comprehends the progress of epidemiological transition in India.

Data and methods

Data. The age-cause-specific death rates (ACSDR) for 21 causes of death (level 2) by quinquennial age groups up to 95 + years and sex were retrieved from Global Burden of Disease (GBD)⁴⁶ for the entire period of 1990–2019 (Supplementary Figs. S1 and S2). The causes of death are mapped with International Classification of Diseases (ICD) 10 classification⁴⁷ in GBD data. There has been concern and issues for the availability of data and quality of data on causes of death for many countries as obstacles for computing mortality estimates^{48–51}. The GBD data provides mortality estimates adjusted for systematic biases or inaccurate reporting for many countries and is also comparable across regions and time.

Methods. Construction of abridged life tables. Abridged life tables were constructed using the Chiang method^{52,53}, based on five-year moving average of ACSDR of 21 causes of death and overall mortality rate, by sex, in the studied period. Chiang method is based on the derivation of relation for the total number of person-years lived between exact ages x and x + n ($_nL_x$) in terms of the average number of years lived by an individual of age x who dies in the interval (x, x + n) ($_na_x$). The columns of the life table are obtained using the following formulas: $_nq_x$: probability of dying between age x and x + n

$${}_{n}\mathbf{q}_{\mathbf{x}} = \frac{n * ({}_{n}\mathbf{M}_{\mathbf{x}})}{1 + (n - {}_{n}\mathbf{a}_{\mathbf{x}}) * {}_{n}\mathbf{M}_{\mathbf{x}}}$$

 l_x : number of people alive at the exact age *x* among a hypothetical birth cohort of 100,000, usually called the radix of the life table.

$$l_{x+n} = l_x * (1 - {}_n q_x)$$

 $_{n}$ d_x : number of deaths in the age interval *x* to *x* + *n*

$$_{n}d_{x} = l_{x} * _{n}q_{x}$$

 ${}_{n}L_{x}$: total number of person-years lived between exact ages x and x + n

$$_{n}\mathbf{L}_{\mathbf{x}} = n * (\mathbf{l}_{\mathbf{x}} - _{n}\mathbf{d}_{\mathbf{x}} + _{n}\mathbf{a}_{\mathbf{x}} * _{n}\mathbf{d}_{\mathbf{x}})$$

 $_{n}a_{x}$: average number of years lived in the age interval x to x + n

$${}_{n}a_{x} = \frac{{}_{n}\mathbf{L}_{x} - \mathbf{n} * l_{x+n}}{l_{x} - l_{x+n}}$$

 T_x : total number of person-years lived beyond Age x

$$\mathbf{T}_{\mathbf{x}} = \mathbf{T}_{\mathbf{x}+\mathbf{n}} + {}_{n}\mathbf{L}_{\mathbf{x}}$$

ex: average number of years of life remaining for a person alive at the beginning of age interval x.

$$e_x = \frac{T_x}{l_x}.$$

Inequality in age at death. The Gini coefficient at birth (G_0) and the disparity in lifespan (e^{\dagger}) are often applied in the field of demography. G_0 measures the variability in age at death. Whereas, e^{\dagger} measures life years lost due to deaths. Wilmoth and Horiuchi⁵⁴ and Vaupel, et al.³² have shown that many inequality measures, including the Gini, are highly correlated. We applied G_0^{22} as a measure of inequality in age at death because it satisfies the four fundamental properties, i.e. the Pigou-Dalton transfer principle, scale invariance, population variance, and symmetry for an inequality measure²³, and so, is a preferred measure of inequality. The G_0 for an abridged life table is expressed as:

$$G_0 = 1 - \frac{1}{e_0 * [l_0]^2} \sum_{x=0}^{w-1} n * \left[(l_{x+n})^2 + \hat{a}_x \left((l_x)^2 - (l_{x+n})^2 \right) \right]$$

where,

$$\hat{a}_x = \frac{1 - \frac{2}{3}q_x + C_x \left(2 - q_x - \frac{6}{5}C_x\right)}{2 - q_x}$$

$$c_x = a_x - \frac{1}{2}$$
$${}_1\hat{a}_0 = {}_1a_0 \left(1 - {}_1q_0 \frac{3 + 0.831 * {}_1a_0}{2 + {}_1q_0} \right)$$

 \hat{a}_x is the adjusted a_x for deviation in the pace of ${}_nq_x$ by age, and $a_x \left[= \frac{(nL_x/n) - l_{x+n}}{(l_x - l_{x+n})} \right]$ is the person-years lived by the individuals who have died within the given interval.

Decomposition of e_0 and G_0 using the replacement method. Yadav, et al.⁵⁵ shows that discrete⁵⁶ and replacement⁵⁷ methods of decomposition analyses^{23,58} produce similar results for e_0 and G_0 . The difference of quantities between two populations is shown as

$$\Delta e_0, \Delta G_0 = \sum_{i=0}^{n-1} \left(\in_{0, x_{i+1}} - \in_{0, x_i} \right) = \sum_{i=0}^n \in_i$$

and,

$$\epsilon_i = e_0[M^{(x_i)}] - e'_0[M^{(x_i)}], G_0[M^{(x_i)}] - G'_0[M^{(x_i)}].$$

where, $M^{(x_i)}$ is a vector of age-specific death rates (ASDR) with elements m'_x for $x < = x_i$ and m_x for $x > = x_i$.

Decomposition of e_0 and G_0 by causes of death and age groups. Specifically, the contribution of *j*th cause of death to the contribution \in_i in *i*th age-interval [x, x + n) is calculated as

$$\epsilon_{i}^{j} = \left(\frac{{}^{a}m_{x_{i}|x_{i+n}}^{j} - {}^{b}m_{x_{i}|x_{i+n}}^{j}}{{}^{a}m_{x_{i}|x_{i+n}} - {}^{b}m_{x_{i}|x_{i+n}}}\right) \epsilon_{i}$$

where, ${}^{a}m_{x_{i}|x_{i+n}}^{j}$ and ${}^{b}m_{x_{i}|x_{i+n}}^{j}$ are ACSDR of *jth* cause of death in *ith* age-interval [x, x + n) in the population *a* and *b*, respectively, and ${}^{a}m_{x_{i}|x_{i+n}}$ and ${}^{b}m_{x_{i}|x_{i+n}}$ are ASDR in *i*th age-interval [x, x + n) in the population *a* and *b*, respectively. The decomposition analysis provides age-cause-specific contributions to Δe_{0} and ΔG_{0} for both sexes, which are comparable across population subgroups and over time. The age group of 85+ years is the last age group presented for any level of presentation. Also, we have considered neonatal disorders and maternal disorders as separate diseases, so 22 causes of death in total.

Ethical approval. We confirm that all methods were carried out in accordance with relevant guidelines and regulations.

Results

Age-specific contributions to Δe_0 and ΔG_0 , India, 1990–2019. The e_0 for men and women increased from 59.8 and 60.8 years in 1990–1994 to 68.8 and 71.3 years, respectively, during 1990–2019. Contemporaneously, the G_0 values for men and women declined respectively from 0.219 and 0.225 in 1990–1994, 0.188 and 0.181 in 2003–2007, and to 0.154 and 0.146 in 2015–2019 (Fig. 1). Men compared to women show higher G_0 values since the mid-2000s. A higher G_0 value confirms high uncertainty in age at death in men than in women. The decomposition analysis of these G_0 values by quinquennial age groups reveals a huge equalising contribution of 78.1 and 69.6% to ΔG_0 (Fig. 3, Table 1) by male and female in 0–4 years, respectively, which is approximately one-and-a-half times than that to Δe_0 (Fig. 2, Table 1). It testifies that decline in G_0 values is majorly driven by mortality reductions among infants and children.

However, men and women in their adult age group of 15–49 years have shown most dramatic temporal changes from almost *zero* or negative contribution in the late 2000s and then manoeuvre it to considerable values of 19.4 and 25.4% to ΔG_0 (Fig. 3) and 15 and 18.8%, respectively, to Δe_0 in the late 2010s (Fig. 2). This skimpy, meagre contribution of adult ages in men has played a major role for the sex differentials in e_0 and G_0 negatively skewed towards men.

The discordance between age-specific contributions to Δe_0 and ΔG_0 are apparent in the middle (50–69 years) and old (70 + years) age groups. The contribution of 19.5 and 14%, respectively, to Δe_0 by men and women in middle age group is approximately three-folds than that to ΔG_0 . It shows a quantum leap for Δe_0 but not for ΔG_0 . On the other hand, a disequalising (negative) contribution of – 14.5 and – 12.9% to ΔG_0 by men and women in old ages, respectively, signify a disparate role not manifested in the decomposition analysis of Δe_0 (Table 1). These large disequalising contributions impede the progression in G_0 , hence the phenomenon of high e_0 and low G_0 is restrained.

The scrutinization of outputs reveals greater, significant role of infant and child age groups for changes in G_0 and e_0 for the entire period of 1990–2019; however, to take note, their age-specific contributions to ΔG_0 and Δe_0 has more or less remained unchanged over time. Rather, temporal changes in age-specific contributions at adult (15–49 years) especially in men are conspicuous. Further, the middle and old age groups contributed variably to Δe_0 and ΔG_0 , but, their temporal contribution has moderately changed. In sum, India's *pattern* of age-specific

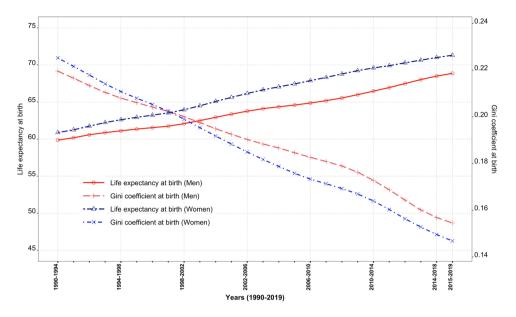


Figure 1. Trends in e_0 and G_0 , men and women, 1990–2019.

	Δe_0		ΔG_0		
Age groups	Men (9 years)	Women (10.5 years)	Men (- 0.065)	Women (- 0.079)	
0-1	27.7	21.9	49.5	36.8	
1-4	16.5	20.1	28.6	32.8	
5-9	4.8	5.8	8.1	9.3	
10-14	1.9	2.2	3.1	3.4	
15-19	2.6	3.4	4.1	5.2	
20-24	2.3	4.0	3.5	5.8	
25-29	1.8	2.9	2.6	4.1	
30-34	1.7	2.6	2.2	3.4	
35-39	1.3	2.2	1.6	2.7	
40-44	2.1	1.9	2.4	2.1	
45-49	3.3	1.9	3.1	1.9	
50-54	3.8	1.5	2.8	1.3	
55-59	3.5	3.2	1.7	2.1	
60-64	6.0	4.0	1.2	1.6	
65-69	6.2	5.3	- 1.0	0.4	
70-74	5.8	5.8	- 3.2	- 1.8	
75-79	4.7	5.8	- 4.5	- 4.2	
80-84	2.5	3.3	- 3.3	- 3.8	
85+	1.6	2.3	- 2.5	- 3.2	

Table 1. Age-specific per cent contributions to Δe_0 and ΔG_0 , men and women, India, 1990–1994 and 2015–2019. Source: Own calculations.

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contributions to Δe_0 or ΔG_0 has remained more or less similar during studied period; nonetheless, the *pattern* has shown encouraging mortality changes at adult ages since the mid-2000s.

Dominance of chronic NCDs versus CDs, India, 1990–2019. The decomposition analysis by causes of death demonstrate that communicable diseases (CDs), NCDs, and injuries contributed respectively 85.6, 4.6, and 9.8% in men and 86.8, 4.8, and 8.3% in women to ΔG_0 between 1990–1994 and 2015–2019 (Table 2). The contributions to ΔG_0 by NCDs were larger when compared to that of Δe_0 . The results show that CDs committedly reshapes the distribution of age at death, whereas NCDs hardly matters for equalising age at death. Exploring by 22 causes of death (Figs. 4 and 5) reveals that the huge contribution by CDs in men and women comprises of the largest contribution of 24.4 and 20.9%, respectively, to ΔG_0 by respiratory infections and tuberculosis. Respiratory infections and tuberculosis together with other infectious diseases, enteric infections, and neonatal

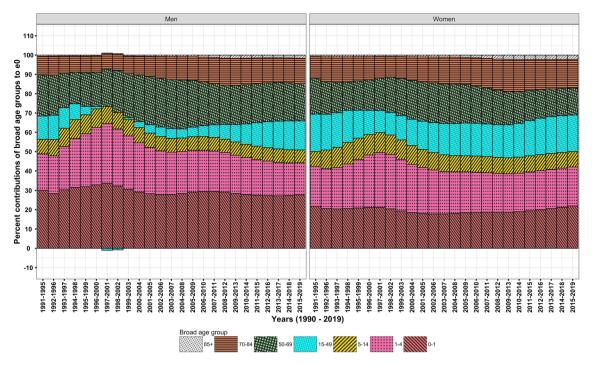


Figure 2. Temporal changes in age-specific contributions to Δe_0 , men and women, 1990–2019.

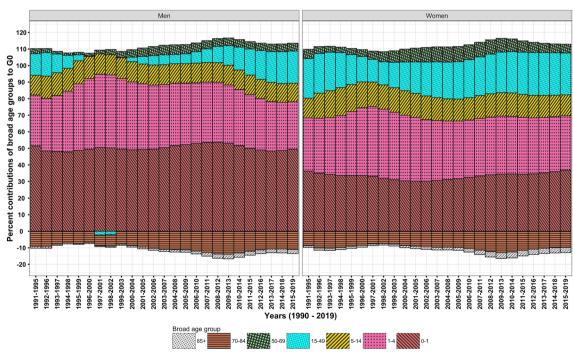


Figure 3. Temporal changes in age-specific contributions to ΔG_0 , men and women, 1990–2019.

and maternal disorders have contributed approximately 72.7 and 74.1% to ΔG_0 in men and women, respectively. Temporal analysis reveals that the share of these four diseases have remained nearly stagnant to ΔG_0 (Fig. 5) and Δe_0 (Fig. 4) in the studied period. This nearly stagnant contributions confirm a consistent mortality decline for these four diseases. In addition to that, the share of neonatal disorders in female children has been approximately doubled from 6.9 in 1990–1994 to 11.4% to ΔG_0 in 2015–2019, confirming a mortality decline among female children. However, the share of neglected tropical diseases and malaria and nutritional deficiencies reduced by half to ΔG_0 during 1990–2019 because of mortality increase. Of great importance, these CDs manoeuvre a low dispersion in the distribution of age at death and guides the causes of death structure.

	Causes of death	Δe_0		ΔG_0	
Broad category		Men (9 years)	Women (10.5 years)	Men (- 0.065)	Women (- 0.079)
CDs	Enteric (Diarrhea and Typhoid) infections	19.0	26.6	14.7	17.1
CDs HIV/AIDS and sexually transmitted infections		- 0.6	- 0.5	- 0.5	- 0.5
CDs	Neonatal disorders	9.0	6.9	15.8	11.4
CDs	Maternal disorders	-	4.5	-	6.2
CDs	Neglected tropical diseases and malaria	3.2	3.2	5.8	5.4
CDs	Nutritional deficiencies	5.3	6.1	7.5	7.9
CDs	Other infectious diseases	11.4	12.2	17.8	18.4
CDs	Respiratory infections and tuberculosis	24.1	19.1	24.4	20.9
Communicable disease	es (CDs)	71.5	78.2	85.6	86.9
Inj	Self-harm and interpersonal violence	1.9	2.4	2.3	3.2
Inj	Transport injuries	0.9	0.7	1.3	0.9
Inj	Unintentional injuries	5.3	3.8	6.2	4.2
Injuries (Inj)		8.1	6.9	9.8	8.3
NCDs	Cardiovascular diseases	7.7	6.2	0.0	0.6
NCDs	Chronic respiratory diseases	6.4	4.8	- 1.3	- 0.4
NCDs	Diabetes and kidney diseases	- 0.2	- 0.2	0.0	0.1
NCDs	Digestive diseases	3.8	2.4	1.9	1.4
NCDs	Mental disorders	0.0	0.0	0.0	0.0
NCDs	Musculoskeletal disorders	0.0	0.0	0.0	0.0
NCDs	Neoplasms	0.5	- 0.1	0.4	0.4
NCDs	Neurological disorders	0.4	0.3	0.7	0.6
NCDs	Other non-communicable diseases	1.8	1.4	2.8	2.1
NCDs	Skin and subcutaneous diseases	0.1	0.1	0.1	0.1
NCDs	Substance use disorders	0.1	0.0	0.0	0.0
Noncommunicable dis	seases (NCDs)	20.6	14.9	4.6	4.9

Table 2. Per cent contributions of causes of death to Δe_0 and ΔG_0 , men and women, India, 1990–1994 and 2015–2019. Source: Own calculations.

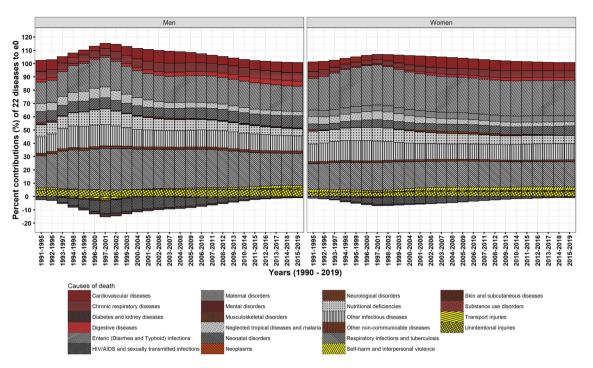
HIV/AIDS and sexually transmitted infections is the only disease among CDs, which had shown negative contribution to Δe_0 as well as disequalising contribution to ΔG_0 for more than 20 years between the mid-1990s and the mid-2010s. Nonetheless, this negative contribution of HIV/AIDS and sexually transmitted infections reduced to ~ – 0.5% in 2015–2019, with the rapid decline in its prevalence and mortality rate.

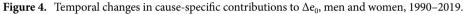
Among injuries, unintentional injuries in men and women have shown small contributions of 6.2 and 4.2% to ΔG_0 , respectively. Other two causes of death, namely self-harm and interpersonal violence and transport injuries, showed very small contributions (Table 2).

Compared to CDs and injuries, NCDs have shown small contribution to Δe_0 and negligible contribution to ΔG_0 . Chronic respiratory diseases, mainly comprise of chronic obstructive pulmonary disease (COPD), asthma, and pneumoconiosis, and cardiovascular diseases, mainly comprise of hypertensive heart disease and strokes, have shown contributions in the range of 6–8% to Δe_0 ; however, they showed negligible contributions to ΔG_0 . Specifically, chronic respiratory diseases and cardiovascular diseases in men has shown disequalising contributions to ΔG_0 between the mid-2000s to the mid-2010s which indicates expansion of deaths over age and time.

In sum, NCDs shows insignificant effect on the dispersion in age at death besides small contribution to Δe_0 that corroborates a shift in age at death. Negligible contributions by many causes of death among NCDs to ΔG_0 intrinsically negate their small contributions to Δe_0 . Thus, NCDs has shown an insufficient contribution and attests its aftermath by impeding the progression in G_0 . Further, the dominance of NCDs at adult and higher ages is also upheld by the modest adult-, middle-, and old-age mortality decline.

Transformation in distribution of age at death by causes of death, India, 1990–2019. India during the period of 1990–2019 shows an impressive pattern of age-specific contributions mainly dominated by CDs in infant through old ages (Figs. 6, 7, 8, and 9). Infants show reduction in their toll of deaths caused by neonatal disorders which have been most crucial for a shorter toe of the tilted *j*-shaped age pattern of mortality (see Supplementary Figs. S1 and S2) and thus, to a more left-skewed unimodal distribution of age at death. This transformation was also sustained by the reduction in the burden caused by respiratory infections and tuber-





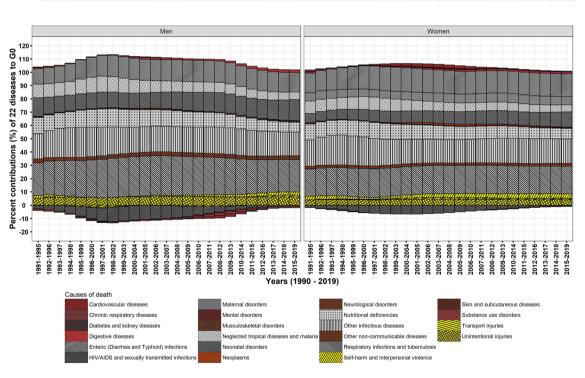


Figure 5. Temporal changes in cause-specific contributions to ΔG_0 , men and women, 1990–2019.

culosis, enteric infections, other infectious diseases, and nutritional deficiencies in infant as well as child and adolescent age groups (Figs. 7 and 9).

In the later stages of life, i.e. from adult to old ages, respiratory infections and tuberculosis and enteric infections show their distinguishable contributions to Δe_0 and ΔG_0 (Figs. 6 and 8). Adult men and adult women show respectively 4.5 and 9.2% whereas middle-aged men and middle-aged women show 2.3 and 3.7% reduction in toll of deaths caused by respiratory infections and tuberculosis and enteric infections for a better G_0 (Figs. 8 and 9). Adult women did experience an aversion of 6.5% maternal deaths in addition to whatsoever reckoned in adult men (Figs. 7 and 9). Contrary to these equalising effects by many CDs, HIV/AIDS and sexually transmitted

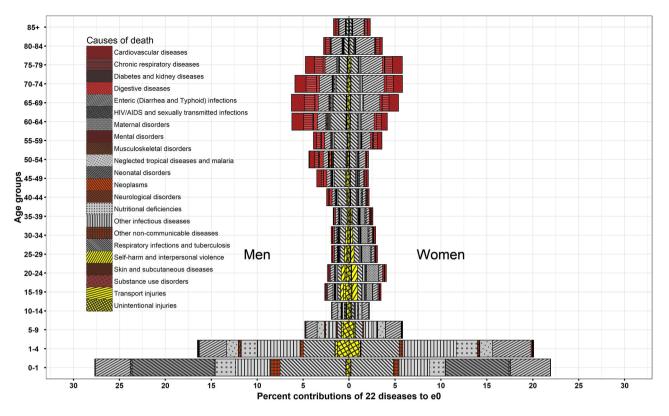


Figure 6. Age-cause-specific contributions to Δe_0 , men and women, between 1990–1994 and 2015–2019.

diseases exhibited disequalising effect on ΔG_0 by adult men and adult women which lessens the equalising contribution in adult age group for a span of time (Fig. 9).

Men and women in their middle-age group have shown small contributions of 2.3 and 3.7% to ΔG_0 , respectively, by respiratory infections and tuberculosis together with enteric infections. Compared to these two predominant diseases, cardiovascular diseases plus chronic respiratory diseases for men and women respectively have shown smaller contribution of 0.8 and 1.7% in adult age group and 1.1 and 1.8% in the middle age group to ΔG_0 (Figs. 7 and 9). In sum, these four causes of death contributed on an average 3.3 and 5.5% to ΔG_0 (Fig. 9) whereas 17.8 and 14.6%, respectively, to Δe_0 (Fig. 7) by men and women in their middle ages. These large contribution to Δe_0 by enteric infections and respiratory infections and tuberculosis at middle ages reveals negligible changes in the distribution of age at death. The middle-aged persons survived and delayed their deaths; thus, they have been instrumental for a shift in the distribution of age at death by adding a few points to e_0 but insignificantly contribute to its dispersion. Thus, its insufficient contribution for a change in dispersion curbs the progression in G_0 .

The progression in G_0 is further opposed by the three-fold disequalising effect for both men and women in their old age group of 70 + years in comparison to that of middle age group by the same causes of death, i.e. respiratory infections and tuberculosis, enteric infections, cardiovascular diseases and chronic respiratory diseases. By causes of death, the disequalising effect in 70 + years is mainly contributed by enteric infections; on an average, a contribution of -4.2 and -6.4% to ΔG_0 in men and women, respectively, during 1990–2019 (Fig. 9). The disequalising effect in old men and old women were approximately -1.5 and -2% to ΔG_0 , respectively, for each of cardiovascular diseases, chronic respiratory diseases, and respiratory infections and tuberculosis and enteric infections (Fig. 9). The disequalising contributions by these dominant causes of death at old ages are a consequence of a higher mortality rates with minor changes at old ages (Figs. 8 and 9). Also, disequalising contribution at old ages in India remains large because of senility and low threshold age.

To note, the chronic NCDs, enteric infections and respiratory infections and tuberculosis showed larger disequalising contributions in old age group than small equalising contributions in the middle age group. So, the net contribution by chronic NCDs and enteric infections and respiratory and infectious diseases in higher age groups is negative. Also, other causes of death showed negligible contributions in middle and old age groups. It implies that a wide age-interval comprising middle and old ages in India is not at all contributing to the progression in G_0 .

The outcomes reveal that the transformations in age at death are majorly contributed by enteric infections followed by respiratory diseases and infectious diseases. CDs have been contributory for a shift in e_0 as well as reshaping the distribution of age at death despite its lower mortality rate than that of NCDs during the studied period. Contrary to the remarkable role of CDs, many NCDs did not contribute enough to equalising age at death in adult through old ages; however, they corroborate for a trivial shift in e_0 . Nonetheless, a greater change in CDs and a subtle change in NCDs testify the progression in the later phases of epidemiological transition.

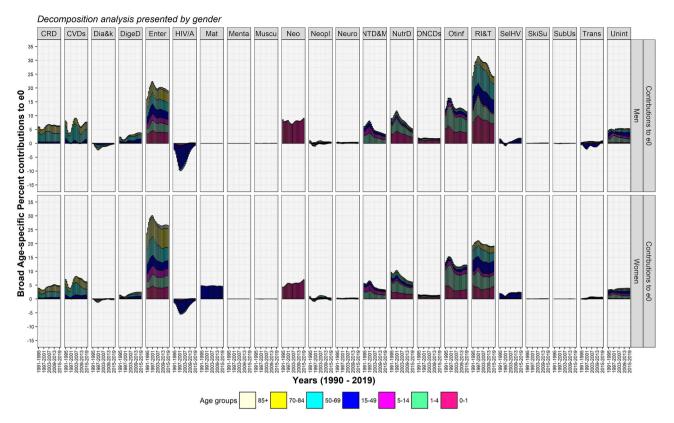


Figure 7. Temporal changes in age-cause-specific contributions to Δe_0 , men and women, India, 1990–2019. *CRD*, chronic respiratory diseases, *CVDs* cardiovascular diseases, *Dia&* diabetes and kidney diseases, *DigeD* digestive diseases, *Enter* enteric (diarrhea and typhoid) infections, *HIV/A* HIV/AIDS and STI, *Mat* maternal disorders, *Menta* mental disorders, *Muscu* musculoskeletal disorders, *Neo* neonatal disorders, *Neopl* neoplasms, *Neuro* neurological disorders, *NTD&* meg. tropical diseases and malaria, *NutrD* nutritional deficiencies, *ONCDs* other noncommunicable diseases, *Otinf* other infectious diseases, *RI&* respiratory infections and tuberculosis, *SelHV* self-harm and interpersonal violence, *SkiSu* skin and subcutaneous diseases, *SubUs* substance use disorders, *Trans* transport injuries, *Unint* unintentional injuries.

Overall, the results reveal a worsening phase in epidemiological transition caused by chronic NCDs in adult, middle, and old age groups, with nearly unchanged toll of deaths caused by injuries in adult ages, with great contribution of mortality decline of CDs in infant through oldest of old ages. While the analyses of e_0 conceals the reason for modest changes in cause of death structure, particularly for NCDs, the analyses of G_0 apparently highlights negligible contribution in wide age-interval in middle through old ages. The outcomes unravel that progression in epidemiological transition is curbed by the high inequality in age at death caused by chronic NCDs in India. Higher inequalities in age at death contributed by the mortality pattern of chronic NCDs raises concern about the structural changes in causes of death not befitting the progression in epidemiological transition.

Discussion

India transcended from the high mortality regime in the early 1990s to a low mortality regime in the late 2010s. Despite showing a significant decline in the burden of CDs mainly among infants and children during 1990–2019, the adults and olds are enduring the heavy burden of NCDs together with that of CDs. There are evidences of chronic NCDs contributing for a rise in e_0 (Figs. 2 and 4); nonetheless, its role for causes of death structure explicitly examined by disparity in lifespan is more crucial. In particular, for India, mortality analysis by causes of death has largely remained unexplored. Acknowledging the gap, this study examines the inequality in mortality (Fig. 1) by 22 causes of death by performing the decomposition analyses⁵⁹ to assess age-cause-specific contributions to the changes in life expectancy at birth (Δe_0) and inequality in age at death (ΔG_0), using GBD data for the entire period of 1990–2019. The study aims to examine the progress of epidemiological transition during a period of 30 years between 1990 and 2019.

The study outcomes reveal that many CDs rather than NCDs significantly contributed to Δe_0 and ΔG_0 (Table 2). A reduction in the burden of CDs in infant through old ages signify their greater role for a structural change in causes of death (Figs. 4 and 5). Amongst demographic age groups, infants have been the largest contributor to Δe_0 and ΔG_0 followed by children, adolescents, and women in their reproductive age groups (Figs. 2 and 3). The same is also corroborated by the rapid decline in infant mortality rate and under-five mortality (U5MR), and Maternal Mortality Ratio (MMR)⁶⁰ over time. Further, the dramatic changes in the age-specific contributions by men in adult age group is bolstered by the reduction in the burden of HIV/AIDS and sexually

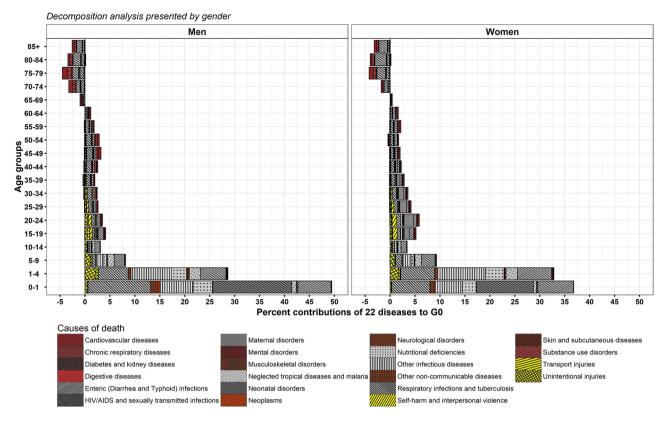


Figure 8. Age-cause-specific contributions to ΔG_0 , men and women, between 1990–1994 and 2015–2019.

transmitted diseases⁶¹ together with that of respiratory infections and tuberculosis and enteric infections. Enteric infections and respiratory infections and tuberculosis also show considerable contributions to Δe_0 and ΔG_0 at middle and old ages (Figs. 7 and 9).

However, many NCDs at middle through old ages contribute considerably to Δe_0 but negligibly to ΔG_0 . The disparateness in the age-specific contributions to Δe_0 (Figs. 6 and 7) and ΔG_0 (Figs. 8 and 9) by CDs and NCDs at middle and old ages highlights the contrasts for a shift in e_0 versus transformation in age at death as measured by G_0 . The mortality decline for CDs at infant through old ages showed a shift in e_0 as well as reshaped the distribution of age at death; however, the mortality decline for NCDs at middle and old ages trivially corroborated the shift in e_0 but importantly didn't contribute to the transformation in age at death or its dispersion (G_0) (Figs. 6, 7, 8, and 9). A negligible contribution to ΔG_0 by NCDs at middle through old ages was on the account of its disequalising contributions.

By the virtue of equalising (positive) and disequalising (negative) effects on G_0 , the NCDs and CDs showing affirmative contributions before the threshold age, however, do not contribute for a better G_0 after that threshold age^{17,62} (Figs. 8 and 9). The low threshold age in 65–69 years⁶² is critical for India (Fig. 9) because a possibility of reduction in premature deaths is restrained by a narrow age-interval. On the other hand, a wide age-interval at middle through old ages burdened with NCDs as well as CDs allows for high disparity in lifespan. Thus, a low threshold age puts major constraints for possible affirmative age-specific contributions from NCDs emplaced at middle and old ages. The same is also applicable for CDs, however, given their preponderance at infant through old ages they do have large reduction in the burden at younger ages that compensates for disequalising contributions in old ages. Such changes in mortality rates by age and variations in causes of death has a repercussion on the age pattern of mortality. While there are significant changes in the mortality rates and variations in cause of death over time in the infant, child, and adult age groups; however, the same is modest in middle through old ages which is also evident in terms of modest mortality deceleration at old ages over time^{42,63}. As a consequence, the *pattern* of age-specific contributions has remained more or less unchanged and a modest structural change in causes of death is witnessed in India.

On the other hand, developed countries such as Japan, Sweden, Switzerland, Singapore, Australia, Germany, Russia, the USA, and other developed countries show significant changes in the *pattern* of age-specific contributions as well as causes of death structure, and importantly high causes-of-death variation^{23,64–66}. Bergeron-Boucher et al.¹³ for low-mortality countries demonstrate significant rise in cause-of-death variation, measured by entropy, since the early 1990s. Yoshinaga and Une⁶⁷ for Japan demonstrate dramatic changes in the cause of death structure from the dominance of tuberculosis and pneumonia until the 1960s to cerebrovascular diseases between 1970 and 1990s, and heart diseases other than ischemic heart disease since 2000s, along with shift in the major contributing age group, i.e. from 0–4 years to 75–84 years. Japan manifests changes in causes of death structure many times and swift changes in the *pattern* of age-specific contributions to gain one of the highest e₀ in the world. Denmark show a significant change in the contribution of middle age group which was negligible during

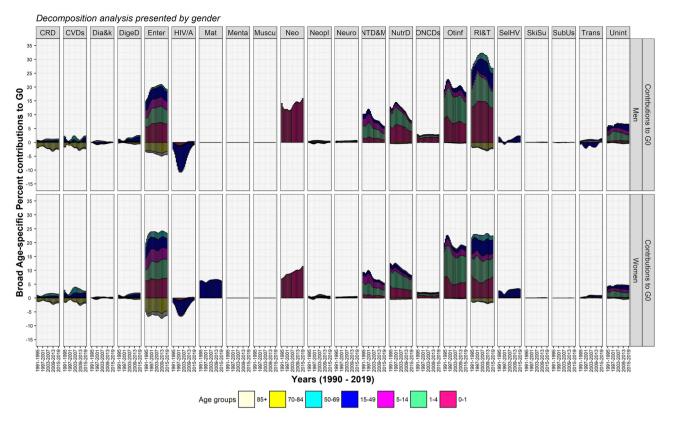


Figure 9. Temporal changes in age-cause-specific contributions to ΔG_0 , men and women, India, 1990–2019. *CRD*, chronic respiratory diseases, *CVDs* cardiovascular diseases, *Dia&k* diabetes and kidney diseases, *DigeD* digestive diseases, *Enter* enteric (diarrhea and typhoid) infections, *HIV/A* HIV/AIDS and STI, *Mat* maternal disorders, *Menta* mental disorders, *Muscu* musculoskeletal disorders, *Neo* neonatal disorders, *Neopl* neoplasms, *Neuro* neurological disorders, *NTD&M* neg. tropical diseases and malaria, *NutrD* nutritional deficiencies, *ONCDs* other noncommunicable diseases, *Otinf* other infectious diseases, *RI&T* respiratory infections and tuberculosis, *SelHV* self-harm and interpersonal violence, *SkiSu* skin and subcutaneous diseases, *SubUs* substance use disorders, *Trans* transport injuries, *Unint* unintentional injuries.

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1960–75, and increased to large, significant contribution of ~ 50% during 1995–2014¹⁷. Such mortality changes at middle through old $ages^{64,67,68}$ concomitant of causes of death structure impels a stronger transformation in the distribution of age at death in order to keep epidemiological transition apace in these developed countries^{1,69}.

On the contrary, India lacks such demographic developments. Linear increase in e_0 depicts a smooth progression in mortality transition, however, scrutinization of G_0 reveals high inequality in age at death caused by NCDs, similar *pattern* of age-specific contributions over time, and modest changes in the cause of death structure mainly attributed to CDs in the studied period. Yadav and Arokiasamy⁷⁰ demonstrate a change in the causes of death structure wherein the burden of NCDs surpassed CDs in mid-1980s; however, thereafter, since the early 1990s the age pattern of mortality of many NCDs marginally changed accounting for toll of deaths (Figs. 5 and S1). The results in this study showed high inequality in mortality distribution causes by many NCDs which was camouflaged in the analysis of e_0 . The outcome of the study demonstrates that high inequality mortality caused by many NCDs have been responsible for slowing down the advances in the epidemiological transition during a period of 30 years between 1990 and 2019. Furthermore, the untimely and behindhand programs for chronic NCDs⁷¹ has already instigated morbidity expansion that rather strengthens dual burden of diseases in India.

In particular, India lacks a mortality decline at the middle ages. The mortality decline at middle ages dominated by high mortality rates of chronic NCDs presents three major benefits: (a) large equalising effect for decline in G_0 , (b) shift in the threshold age, and (c) benign disequalising contributions in old ages. This demographic development can be achieved by reduction in toll of deaths caused by enteric infections, respiratory infections and tuberculosis, cardiovascular diseases, and chronic respiratory diseases in the middle age group of 50–69 years. It appalling to note that despite a linear increase in e_0 , India shows a modest reduction in premature mortality that in turn undermines the fundamental demographic processes such as mortality compression, the phenomenon of high e_0 and low G_0 , and morbidity compression. The middle age group provides a compatible possibility for progress of these fundamental demographic processes and epidemiological as well as mortality transition comparable to that in developed countries in a short time. The developed nations demonstrate advances in the third and later phases^{6,7} of epidemiological transition by a greater role of middle age group for structural changes among NCDs, i.e. in the age pattern of mortality, causes of death structure, and importantly causes-of-death variation. The Ministry of Health and Family Welfare (MoHFW) in its recent health report National Multisectoral Action Plan (NMAP) for Prevention and Control of Common Noncommunicable Diseases (2017–2022)⁷², National Programme for Health Care of Elderly (NPHCE)⁷³ and National Programme for Prevention and Control of Cancer, Diabetes, Cardiovascular diseases and Strokes (NPCDCS)⁷⁴ strategizes for the assessment of economic and mortality burden of NCDs by strengthening patient data and harmonization of disease data. Under NPCDCS, the MoHFW has proposed to developed standard protocols for data collection, analysis, and reporting of data for different NCDs services at all levels. For this purpose, the Health Management Information System (HMIS) is also leveraged to identify linkage modalities with NCDs services. The interpretation of the results could be better explained with a harmonized data between other sources of data such as Sample Registration System (SRS), Medical Certification of Causes of death (MCCD), and MoHFW, and other health survey data. However, for India, details of causes of death mapped with ICD classification are not available by age, sex and residence for a long period. Also, the use of detailed GBD data needs cautious interpretations^{50,51,75,76}. While the life table estimates between GBD data and SRS are very close; nonetheless, synchronous details of causes of death since the early 1990s are available in GBD data.

The national NCD monitoring framework in way forward has prioritise the reduction of premature mortality from 10 to 25% in another five years in the age group of 30–70 years caused by cardiovascular diseases and chronic respiratory diseases⁷². The outcomes of this study point out that surveillance of population in the middle age group of 50–69 years for NCDs, especially COPD, asthma, and pneumoconiosis, can reduce premature mortality in a short time. Also, screening and diagnosis in a narrow age interval lessens the burden on public health system and ascertain effective utilization of limited sources. The contribution of middle ages to mortality decline has been undermined just because of low threshold age in India. This remained neglected since a long time in the policy frameworks as well as research studies. The MoHFW need to prioritise for the surveillance and investment in the middle age group of 50–69 years which promises high possibility to reduction in the premature mortality.

Conclusion

The study reveals (1) large, significant contributions of CDs for reshaping the distribution of age at death and (2) an exacerbated predicament offset by the intrusion of NCDs for a high inequality in age at death. During a period of 30 years between 1990–1994 and 2015–2019, the structural changes in causes of death has been attributed to CDs and marginally to NCDs. A subtle contribution of NCDs to the transformation in distribution of age at death is evident.

The progression in epidemiological transition is modulated mainly by two factors: (a) moderate contribution of adult age group and (b) greater mortality decline attributable to CDs. The role of NCDs is emplaced at middle and higher ages and the pace of epidemiological transition has been modest because of low threshold age in India; importantly, the possible contribution of middle age group is constrained attributable to high inequality in NCDs. The study reveals the high inequality in age at death in India caused by NCDs at middle (50–69 years) ages is the priority to deal effectively in policies and programs. These are underlying reasons for the prolonged phenomenon of dual burden of diseases in India. In a long period of 30 years, India has shown modest changes in causes of death structure. It rather manifests a divergence from that of oversimplified the Omran's epidemiological transition.

Our study outcomes highlight the urgent tuning of policies targeting middle-aged persons as their survival can make a shift in the threshold age, contribute to structural changes in cause of death, and low inequality in age at death. This will lessen the dual burden of diseases in India for a demographic leap, morbidity compression, prevent health losses and increase lifespan spent in good health.

Data availability

The datasets generated and/or analysed during the current study are available in the Global Burden of Disease Study 2019 (GBD 2019), Institute for Health Metrics and Evaluation (IHME), United States, http://ghdx.healt hdata.org/gbd-results-tool⁴⁶.

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References

- 1. Salomon, J. A. & Murray, C. J. L. The epidemiologic transition revisited: compositional models for causes of death by age and sex. *Popul. Dev. Rev.* 28, 205–228. https://doi.org/10.1111/j.1728-4457.2002.00205.x (2002).
- 2. Omran, A. R. The epidemiologic transition. A theory of the epidemiology of population change. *Milbank Mem. Fund. Q* 49, 509–538 (1971).
- Moser, K., Shkolnikov, V. & Leon, D. A. World mortality 1950–2000: Divergence replaces convergence from the late 1980s. Bull. World Health Organ. 83 (2005).
- Santosa, A., Wall, S., Fottrell, E., Hogberg, U. & Byass, P. The development and experience of epidemiological transition theory over four decades: A systematic review. *Glob. Health Action* 7, 23574. https://doi.org/10.3402/gha.v7.23574 (2014).
- 5. Caselli, G., Meslé, F. & Vallin, J. Epidemiologic transition theory exceptions. Genus 58, 9-51 (2002).
- 6. Olshansky, S. J. & Ault, A. B. The fourth stage of the epidemiologic transition: The age of delayed degenerative diseases. *Milbank* Q 64, 355–391 (1986).
- Hazra, N. C. & Gulliford, M. Evolution of the "fourth stage" of epidemiologic transition in people aged 80 years and over: Population-based cohort study using electronic health records. *Popul. Health Metr.* 15, 18. https://doi.org/10.1186/s12963-017-0136-2 (2017).
- Mercer, A. J. Updating the epidemiological transition model. *Epidemiol. Infect.* 146, 680–687. https://doi.org/10.1017/S095026881 8000572 (2018).
- 9. Mercer, A. Infections, Chronic Disease, and the Epidemiological Transition. A New Perspective. (University of Rochester Press, 2014).
- 10. Omran, A. R. The epidemiologic transition theory revisited thirty years later. World Health Stat. Q. 53, 99–119 (1998).

- 11. Robine, J. M. Redefining the Stages of the epidemiological transition by a study of the dispersion of life. *Popul. English Select.* **13**, 173–194 (2001).
- Vos, T. *et al.* Global burden of 369 diseases and injuries in 204 countries and territories, 1990–2019: A systematic analysis for the Global Burden of Disease Study 2019. *The Lancet* 396, 1204–1222. https://doi.org/10.1016/s0140-6736(20)30925-9 (2020).
- Bergeron-Boucher, M. P., Aburto, J. M. & Raalte, A. A. V. Diversification in causes of death in low-mortality countries: emerging patterns and implications. *BMJ Glob. Health* 5. https://doi.org/10.1136/bmjgh-2020-002414 (2020).
- Norheim, O. F. *et al.* Avoiding 40% of the premature deaths in each country, 2010–30: Review of national mortality trends to help quantify the UN Sustainable Development Goal for health. *Lancet* 385, 239–252. https://doi.org/10.1016/S0140-6736(14)61591-9 (2015).
- Leon, D. A., Jdanov, D. A. & Shkolnikov, V. M. Trends in life expectancy and age-specific mortality in England and Wales, 1970–2016, in comparison with a set of 22 high-income countries: An analysis of vital statistics data. *The Lancet Public Health* 4, e575–e582. https://doi.org/10.1016/s2468-2667(19)30177-x (2019).
- Aburto, J. M., Villavicencio, F., Basellini, U., Kjaergaard, S. & Vaupel, J. W. Dynamics of life expectancy and life span equality. Proc. Natl. Acad. Sci. U S A 117, 5250–5259. https://doi.org/10.1073/pnas.1915884117 (2020).
- Aburto, J. M., Wensink, M., Raalte, A. A. V. & Lindahl-Jacobsen, R. Potential gains in life expectancy by reducing inequality of lifespans in Denmark: An international comparison and cause-of-death analysis. *BMC Public Health* 18, 831. https://doi.org/10. 1186/s12889-018-5730-0 (2018).
- Vaupel, J. W. & Romo, V. C. Decomposing change in life expectancy: A bouquet of formulas in honor of Nathan Keyfitz's 90th birthday. *Demography* 40, 201–216. https://doi.org/10.1353/dem.2003.0018 (2003).
- Aburto, J. M., Alvarez-Martínez, J.-A., Villavicencio, F. & Vaupel, J. W. The threshold age of the lifetable entropy. *Demogr. Res.* 41, 83–102. https://doi.org/10.4054/DemRes.2019.41.4 (2019).
- Shkolnikov, V. M., Andreev, E. M., Zhang, Z., Oeppen, J. & Vaupel, J. W. Losses of expected lifetime in the United States and other developed countries: Methods and empirical analyses. *Demography* 48, 211–239. https://doi.org/10.1007/s13524-011-0015-6 (2011).
- van Raalte, A. A. & Caswell, H. Perturbation analysis of indices of lifespan variability. *Demography* 50, 1615–1640. https://doi.org/ 10.1007/s13524-013-0223-3 (2013).
- 22. Hanada, K. A formula of Gini's concentration ratio and its application to life tables. J. Japan Stat. Soc. 13, 95–98 (1983).
- Shkolnikov, V. M., Andreev, E. E. & Begun, A. Z. Gini coefficient as a life table function: computation from discrete data, decomposition of differences and empirical examples. *Demogr. Res.* 8, 305–358. https://doi.org/10.4054/DemRes.2003.8.11 (2003).
- Németh, L. Life expectancy versus lifespan inequality: A smudge or a clear relationship?. PLoS ONE 12, e0185702. https://doi.org/ 10.1371/journal.pone.0185702 (2017).
- Fries, J. F., Bruce, B. & Chakravarty, E. Compression of morbidity 1980–2011: A focused review of paradigms and progress. J. Aging Res. 1–10, 2011. https://doi.org/10.4061/2011/261702 (2011).
- 26. Kannisto, V. Measuring the Compression of Mortality. Demogr. Res. 13. https://doi.org/10.4054/DemRes.2000.3.6 (2000).
- Yadav, S. & Perianayagam, A. Mortality compression and variability in age at death in India. *Compar. Popul. Stud.* 45. https://doi. org/10.12765/CPoS-2020-20 (2020).
- Shiels, M. S. et al. Trends in premature mortality in the USA by sex, race, and ethnicity from 1999 to 2014: An analysis of death certificate data. Lancet 389, 1043–1054. https://doi.org/10.1016/S0140-6736(17)30187-3 (2017).
- Case, A. & Deaton, A. Rising morbidity and mortality in midlife among white non-Hispanic Americans in the 21st century. Proc. Natl. Acad. Sci. USA 112, 15078–15083. https://doi.org/10.1073/pnas.1518393112 (2015).
- Avendano, M. & Kawachi, I. Why do Americans have shorter life expectancy and worse health than do people in other high-income countries?. Annu. Rev. Public Health 35, 307–325. https://doi.org/10.1146/annurev-publhealth-032013-182411 (2014).
- Acciai, F. & Firebaugh, G. Twin consequences of rising U.S. death rates among young adults: Lower life expectancy and greater lifespan variability. Prev. Med. 127, 105793. https://doi.org/10.1016/j.ypmed.2019.105793 (2019).
- 32. Vaupel, J. W., Zhang, Z. & Raalte, A. A. V. Life expectancy and disparity: an international comparison of life table data. *BMJ Open* 1, 1–6 (2011).
- Jha, P. Reliable direct measurement of causes of death in low- and middle-income countries. BMC Med. 12, 19. https://doi.org/10. 1186/1741-7015-12-19 (2014).
- 34. Kumar, B. G. Low mortality and high morbidity in Kerala reconsidered. Popul. Dev. Rev. 19, 103-121 (1993).
- Sole-Auro, A. & Alcaniz, M. Are we living longer but less healthy? Trends in mortality and morbidity in Catalonia (Spain), 1994–2011. Eur. J. Ageing 12, 61–70. https://doi.org/10.1007/s10433-014-0317-9 (2015).
- Smits, J. & Monden, C. Length of life inequality around the globe. Soc. Sci. Med. 68, 1114–1123. https://doi.org/10.1016/j.socsc imed.2008.12.034 (2009).
- 37. Clark, R. World health inequality: Convergence, divergence, and development. Soc. Sci. Med. 72, 617-624 (2011).
- Claeson, C., Bos, E. R., Mawji, T. & Pathmanathan, I. Reducing child mortality in India in the new millennium. Bull. World Health Organ. 78, 1192–1199 (2000).
- MoHFW (Ministry of Health and Family Welfare). Reproductive & Child Health Programme Phase II, 8th Joint Review Mission. (Ministry of Health and Family Welfare, New Delhi, 2011).
- Arokiasamy, P. & Yadav, S. Changing age patterns of morbidity vis-à-vis mortality in India. J. Biosoc. Sci. 46, 462–479. https://doi. org/10.1017/S002193201300062X (2014).
- Lee, R. D. Rethinking the evolutionary theory of aging: Transfers, not births, shape senescence in social species. Proc. Natl. Acad. Sci. 100, 9637–9642. https://doi.org/10.1073/pnas.1530303100 (2003).
- 42. Yadav, A., Yadav, S. & Kesarwani, R. Decelerating mortality rates in older ages and its prospects through Lee-Carter approach. *PLoS ONE* 7, 1. https://doi.org/10.1371/journal.pone.0050941 (2012).
- India State-level Disease Burden Initiative Collaborators. Nations within a nation: Variations in epidemiological transition across the states of India, 1990–2016 in the Global Burden of Disease Study. *The Lancet* 390, 2437–2460. https://doi.org/10.1016/S0140-6736(17)32804-0 (2017).
- Yadav, S., Kothavale, A., Yadav, P. K. & Yadav, N. in Collaborative Governance for Sustainable Development of Health and Well-Being: Issues and Perspective Vol. I Non-Communicable diseases (eds Harshad P. Thakur et al.) Ch. 7, 117–134 (2021).
- Jagnoor, J. et al. Childhood and adult mortality from unintentional falls in India. Bull. World Health Organ. 89, 733–740. https:// doi.org/10.2471/BLT.11.086306 (2011).
- 46. Global Burden of Disease Collaborative Network. Global Burden of Disease Study 2019 (GBD 2019) Results. (Institute for Health Metrics and Evaluation (IHME), Seattle, United States of America, 2020).
- Global Burden of Disease Collaborative Network. Global Burden of Disease Study 2019 (GBD 2019) Cause List Mapped to ICD Codes., (Institute for Health Metrics and Evaluation (IHME), Seattle, United States of America, 2020).
- Maher, C. & Ferreira, G. Time to reconsider what Global Burden of Disease studies really tell us about low back pain. Ann. Rheum. Dis. 81, 306–308. https://doi.org/10.1136/annrheumdis-2021-221173 (2022).
- Voigt, K. & King, N. B. Out of alignment? Limitations of the global burden of disease in assessing the allocation of global health aid. *Public Health Ethics* 10, 244–256. https://doi.org/10.1093/phe/phx012 (2017).
- Bhalla, K. & Harrison, J. E. GBD-2010 overestimates deaths from road injuries in OECD countries: New methods perform poorly. Int. J. Epidemiol. 44, 1648–1656. https://doi.org/10.1093/ije/dyv019 (2015).

- Garcia-Basteiro, A. L., Brew, J., Williams, B., Borgdorff, M. & Cobelens, F. What is the true tuberculosis mortality burden? Differences in estimates by the World Health Organization and the Global Burden of Disease study. *Int. J. Epidemiol.* 47, 1549–1560. https://doi.org/10.1093/ije/dyy144 (2018).
- Chiang, C. L. On constructing current life tables. J. Am. Stat. Assoc. 67, 538–541. https://doi.org/10.1080/01621459.1972.10481 245 (1972).
- Schoen, R. Calculating life tables by estimating Chiang's a from observed rates. *Demography* 15, 625–635. https://doi.org/10.2307/ 2061212 (1978).
- Wilmoth, J. R. & Horiuchi, S. Rectangularization revisited: Variability of age at death within human populations. *Demography* 36, 475–495. https://doi.org/10.2307/2648085 (1999).
- Yadav, S., Yadav, P. K. & Yadav, N. Impact of COVID-19 on life expectancy at birth in India: A decomposition analysis. BMC Public Health 21, 1906. https://doi.org/10.1186/s12889-021-11690-z (2021).
- Arriaga, E. E. Measuring and explaining the change in life expectancies. *Demography* 21, 83–96. https://doi.org/10.2307/2061029 (1984).
- 57. Kitagawa, E. M. Standardized comparisons in population research. Demography 1, 296-315 (1964).
- Bergeron-Boucher, M., Ebeling, M. & Canudas-Romo, V. Decomposing changes in life expectancy: Compression versus shifting mortality. *Demogr. Res.* 33, 391–424. https://doi.org/10.4054/DemRes.2015.33.14 (2015).
- Wagner, P. Sensitivity of life disparity with respect to changes in mortality rates. Demogr. Res. 23, 63–72. https://doi.org/10.4054/ DemRes.2010.23.3 (2010).
- 60. ORGI (Office of the Registrar General & Census Commissioner, India). Sample registration system (SRS)-statistical report 2020. (MoHFW, GOI, New Delhi, 2020).
- Jha, P. et al. HIV mortality and infection in India: estimates from nationally representative mortality survey of 1.1 million homes. BMJ 340, c621. https://doi.org/10.1136/bmj.c621 (2010).
- 62. Yaday, S. Progress of inequality in age at death in India: Role of adult mortality. Eur. J. Popul. 37, 523-550. https://doi.org/10.1007/s10680-021-09577-1 (2021).
- 63. Horiuchi, S. & Wilmoth, J. R. Deceleration in the age pattern of mortality at older ages. *Demography* **35**, 391–412 (1998).
- Klenk, J., Keil, U., Jaensch, A., Christiansen, M. C. & Nagel, G. Changes in life expectancy 1950–2010: contributions from age- and disease-specific mortality in selected countries. *Popul Health Metr.* 14, 20. https://doi.org/10.1186/s12963-016-0089-x (2016).
- Beltran-Sanchez, H., Preston, S. H. & Canudas-Romo, V. An integrated approach to cause-of-death analysis: Cause-deleted life tables and decompositions of life expectancy. *Demogr. Res.* 19, 1323. https://doi.org/10.4054/DemRes.2008.19.35 (2008).
- Sudharsanan, N., Aburto, J. M., Riffe, T. & van Raalte, A. Commentary: Large variation in the epidemiological transition across countries: Is it still valuable as a mortality theory?. *Int. J. Epidemiol.* https://doi.org/10.1093/ije/dyac107 (2022).
- Yoshinaga, K. & Une, H. Contributions of mortality changes by age group and selected causes of death to the increase in Japanese life expectancy at birth from 1950 to 2000. Eur. J. Epidemiol. 20, 49–57. https://doi.org/10.1007/s10654-004-9557-x (2005).
- Zarulli, V., Kashnitsky, I. & Vaupel, J. W. Death rates at specific life stages mold the sex gap in life expectancy. Proc. Natl. Acad. Sci. USA 118, 1. https://doi.org/10.1073/pnas.2010588118 (2021).
- 69. Kochanek, K. D., Arias, E. & Bastian, B. A. The effect of changes in selected age-specific causes of death on non-Hispanic white life expectancy between 2000 and 2014. *NCHS Data Brief* 1, 1–8 (2016).
- Yadav, S. & Arokiasamy, P. Understanding epidemiological transition in India. *Glob. Health Action* 7, 23248. https://doi.org/10. 3402/gha.v7.23248 (2014).
- Rajaratnam, J. K. *et al.* Worldwide mortality in men and women aged 15–59 years from 1970 to 2010: A systematic analysis. *Lancet* 375, 1704–1720 (2010).
- 72. MoHFW (Ministry of Health and Family Welfare). National Multisectoral Action Plan for Prevention and Control of Common Noncommunicable Diseases (2017–2022). (MoHFW, GOI, New Delhi, 2017).
- 73. MoHFW (Ministry of Health and Family Welfare). Update on National Programme for Health Care of Elderly. (MoHFW, New Delhi, 2022).
- MoHFW (Ministry of Health and Family Welfare). National programme for prevention and control of cancer, diabetes, cardiovascular diseases & stroke (NPCDCS). (MoHFW, New Delhi, 2013).
- King, C. H. & Bertino, A. M. Asymmetries of poverty: Why global burden of disease valuations underestimate the burden of neglected tropical diseases. *PLoS Negl. Trop. Dis.* 2, e209. https://doi.org/10.1371/journal.pntd.0000209 (2008).
- Seligman, B. J., Cullen, M. R. & Horwitz, R. I. Aging, transition, and estimating the global burden of disease. *PLoS ONE* 6, e20264. https://doi.org/10.1371/journal.pone.0020264 (2011).

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Author contributions

S.Y., A.P. conceptualised the study, S.Y. conducted the analyses, S.Y. prepared tables and figures, and S.Y. wrote the first draft manuscript. S.Y., A.P., S.A.P., and S.A.C. revised the manuscript. All authors read and approved the final manuscript.

Competing interests

The authors declare no competing interests.

Additional information

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