

Ageing by Design: Deriving Mortality Models from Reliability Theory

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Joint work with Xiaobai Zhu

Longevity 21

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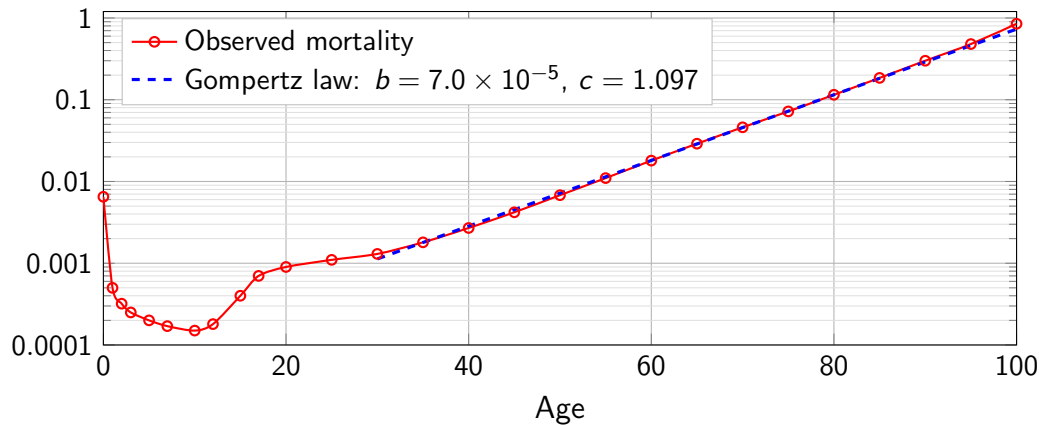
Outline

- 1 Mortality Modelling
- 2 Biological Redundancy
- 3 Mortality Modelling via Biological Redundancy
- 4 Empirical Results
- 5 Discussion

Mortality Modelling

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Mortality Curve



Gompertz Law (Gompertz, 1825)

$$\mu_x = b c^x \iff \ln \mu_x = \ln b + x \ln c$$

Two research questions:

- 1 Why the Gompertz form?
I.e., why the *exponential* growth trend over age?
- 2 Why the Gompertz parameters?
I.e., what are the biological mechanisms behind b and c ?

More Mortality Laws

- *Makeham* (Makeham, 1860)

$$\mu_x = a + b c^x$$

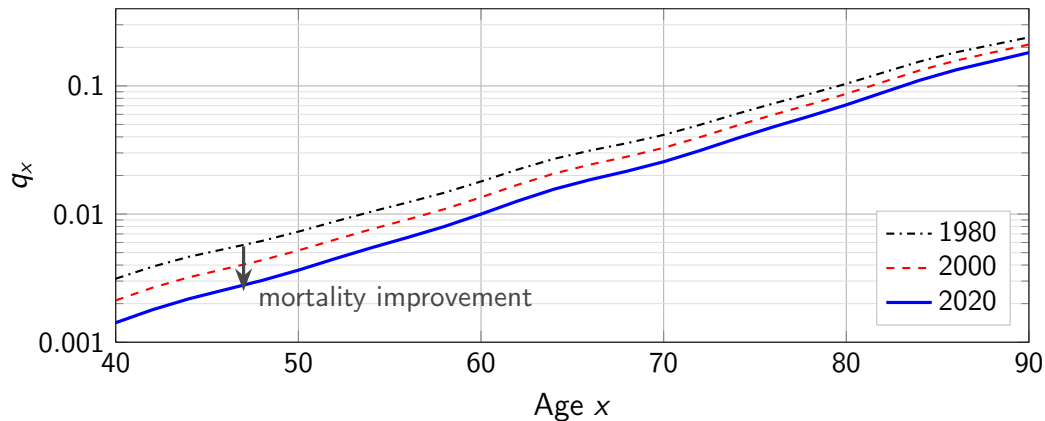
- *Death Plateau* (Perks, 1932)

$$\text{logit } q_x = B + C x$$

- *Heligman-Pollard* (Heligman and Pollard, 1980)

$$\frac{q_x}{1 - q_x} = A^{(x+B)^C} + D e^{-E(\log x - \log F)^2} + G H^x$$

Mortality Curve over Time



Cairns–Blake–Dowd (Cairns et al., 2006)

The CBD model, a stochastic mortality model with two period factors:

$$\text{logit } q_{x,t} = \kappa_t^{(1)} + \kappa_t^{(2)} \cdot (x - \bar{x})$$

where

- $\kappa_t^{(1)}$ captures the improvement in the mortality level;
- $\kappa_t^{(2)}$ captures the change in the ageing speed.

Cohort Effect (Hunt and Blake, 2014)

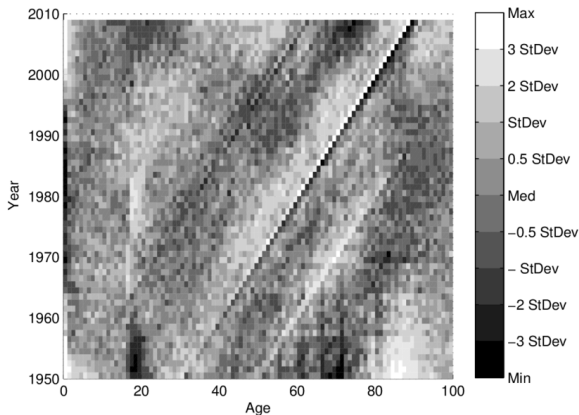


Figure: Residuals of the CBD model, UK Male.

The CBD Family (Cairns et al., 2009)

- The M6 model:

$$\text{logit } q_{x,t} = \kappa_t^{(1)} + \kappa_t^{(2)} \cdot (x - \bar{x}) + \gamma_{t-x}$$

- The M7 model:

$$\text{logit } q_{x,t} = \kappa_t^{(1)} + \kappa_t^{(2)} \cdot (x - \bar{x}) + \kappa_t^{(3)} \cdot [(x - \bar{x})^2 - \hat{\sigma}_x^2] + \gamma_{t-x}$$

- And many other extensions, such as adding economic variables, climate variables, seasonality, etc.

Research Questions

- *Statistical assumptions* over biological mechanisms
E.g., why is mortality logit- or log-linear in age?
- *Interpretability of models* on age, period and cohort effects
E.g., what exactly is a cohort effect?
- *Construction of stress scenarios* on latent factors
E.g., what does a shock to $\kappa_t^{(2)}$ mean?
- *Multi-population extensions* using a top-down approach
E.g., why should two populations share a common factor?

Biological Redundancy

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Reliability Theory of Ageing (Gavrilov and Gavrilova, 2001)

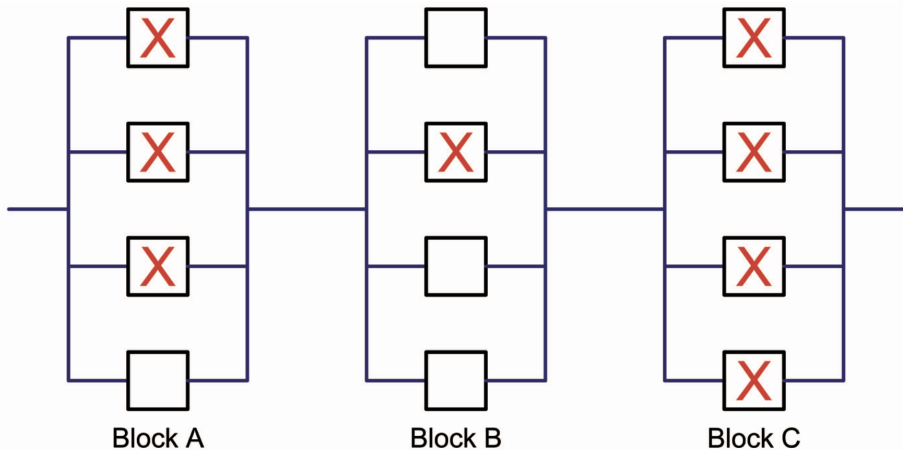


Figure: A Parallel-Series System (Richards, 2008)

Ageing as Decay of Biological Redundancy

Consider an organism composed of N biological subsystems, and let D_x be the number of dysfunctional subsystems at age x .

- We assume mortality is linked to D_x via a link function $h(\cdot)$:

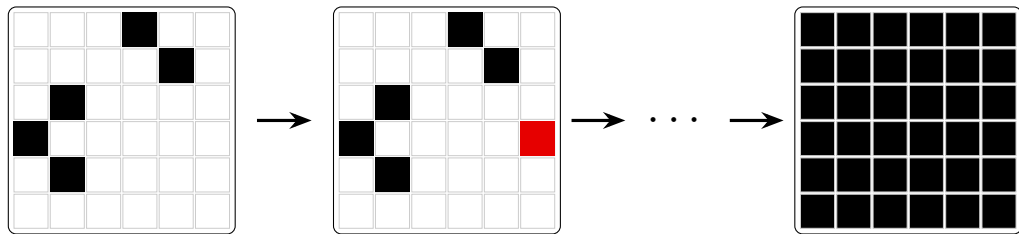
$$\mu_x = h(D_x)$$

- We assume new failures arrive at the rate of

$$g(D_x) \cdot (N - D_x)$$

where $g(D_x)$ is the per-functioning-subsystem failure hazard and $N - D_x$ is the number of subsystems still functioning.

An Example with $N = 36$

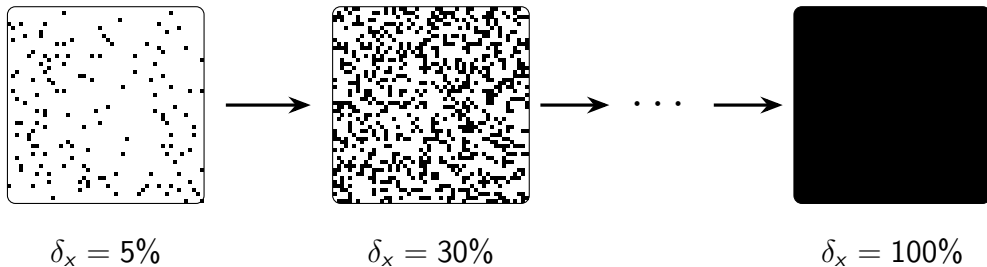


When N is Large

When N is large, we define *failure fraction*:

$$\delta_x = \frac{D_x}{N} \in [0, 1],$$

which is the proportion of subsystems that have failed by age x .



Frailty Index and δ_x

- Frailty Index (FI) from Mitnitski et al. (2001):

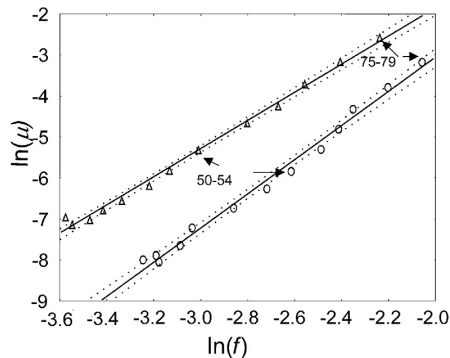
$$FI = \frac{\# \text{ of deficits present}}{\# \text{ of deficits measured}}$$

- Same construction as $\delta_x = \frac{D_x}{N}$; so FI is the *observable counterpart* of δ_x .
- Mitnitski et al. (2002) found that

$$\ln \mu = a + b \ln FI$$

which supports a log-linear link function $h(\cdot)$.

Rate of mortality as a function of frailty index



A Cascade Process

When N is large, the failure fraction satisfies

$$\frac{d\delta_x}{dx} = g(\delta_x)(1 - \delta_x)$$

with initial value δ_{x_0} .

- Consider the per-functioning-subsystem failure hazard (Nielsen et al., 2024)

$$g(\delta) = r\delta$$

where $r \geq 0$ is the ageing rate, and the link function $h(\delta) = \delta$.

- Then, we have

$$\text{logit } \mu_x = \text{logit } \delta_x = \text{logit } \delta_{x_0} + r \cdot (x - x_0)$$

Mortality Modelling via Biological Redundancy

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From Individual to Population

- The parameters δ_{x_0} and r describe a *single organism*: how much damage it starts with, and how fast that damage cascades.
- The ageing mechanism runs along one lifetime; that is, along a *cohort* diagonal with birth year $c = t - x$.
- Parameters that stay constant along that diagonal are exactly parameters indexed by birth *cohort* c .
- Calendar year enters through the *link* function instead: medicine and environment change how damage is recorded as mortality.

CBD/M5 vs R1

Consider $g(\delta) = r\delta$ and $\text{logit } q_x = \text{logit } \delta_x$, then

$$\text{logit } q_x = \text{logit } \delta_{x_0} + r \cdot (x - x_0)$$

Set δ_{x_0} and r to be indexed by *birth cohort* $c = t - x$, then

$$\text{logit } q_{x,t} = \gamma_{t-x}^{(1)} + \gamma_{t-x}^{(2)} \cdot (x - x_0) \quad (\text{R1})$$

where $\gamma_{t-x}^{(1)} = \text{logit } \delta_{x_0, t-x}$ and $\gamma_{t-x}^{(2)} = r_{t-x}$.

In comparison, the CBD/M5 model is

$$\text{logit } q_{x,t} = \kappa_t^{(1)} + \kappa_t^{(2)} \cdot (x - \bar{x}) \quad (\text{M5})$$

M6 vs R2

Consider again $g(\delta) = r\delta$, and add an *age-independent* background term:

$$\text{logit } q_x = \text{logit } \delta_x + \kappa$$

Set κ to be indexed by *year* t , then

$$\text{logit } q_{x,t} = \gamma_{t-x}^{(1)} + \gamma_{t-x}^{(2)} \cdot (x - x_0) + \kappa_t \quad (\text{R2})$$

In comparison, the M6 model is

$$\text{logit } q_{x,t} = \kappa_t^{(1)} + \kappa_t^{(2)} \cdot (x - \bar{x}) + \gamma_{t-x} \quad (\text{M6})$$

Consider a *damage-independent* baseline term in the failure hazard:

$$g(\delta) = \alpha + r\delta$$

Then,

$$\text{logit } \delta_x = \ln \left[\frac{\alpha + r\delta_{x_0}}{(\alpha + r)(1 - \delta_{x_0})} e^{(\alpha+r)(x-x_0)} - \frac{\alpha}{\alpha + r} \right]$$

By Taylor expansion around x_0 ,

$$\text{logit } \delta_x \approx \text{logit } \delta_{x_0} + \frac{\alpha + r\delta_{x_0}}{\delta_{x_0}} \cdot (x - x_0) - \frac{1}{2}\alpha \cdot \frac{\alpha + r\delta_{x_0}}{\delta_{x_0}} \cdot \frac{1 - \delta_{x_0}}{\delta_{x_0}} \cdot (x - x_0)^2$$

M7 vs R3 (Cont.)

Consider again

$$\text{logit } q_x = \text{logit } \delta_x + \kappa$$

Then,

$$\text{logit } q_{x,t} = \gamma_{t-x}^{(1)} + \gamma_{t-x}^{(2)} \cdot (x - x_0) + \gamma_{t-x}^{(3)} \cdot (x - x_0)^2 + \kappa_t \quad (\text{R3})$$

In comparison, the M7 model is

$$\text{logit } q_{x,t} = \kappa_t^{(1)} + \kappa_t^{(2)} \cdot (x - \bar{x}) + \kappa_t^{(3)} \cdot ((x - \bar{x})^2 - \hat{\sigma}_x^2) + \gamma_{t-x} \quad (\text{M7})$$

Model Summary

Model	Linear predictor $\logit q_{x,t}$	# of para	constr.
M5	$\kappa_t^{(1)} + \kappa_t^{(2)} (x - \bar{x})$	$2T$	0
M6	$\kappa_t^{(1)} + \kappa_t^{(2)} (x - \bar{x}) + \gamma_{t-x}$	$2T + A - 2$	2
M7	$\kappa_t^{(1)} + \kappa_t^{(2)} (x - \bar{x}) + \kappa_t^{(3)} ((x - \bar{x})^2 - \hat{\sigma}_x^2) + \gamma_{t-x}$	$3T + A - 3$	3
R1	$\gamma_{t-x}^{(1)} + \gamma_{t-x}^{(2)} (x - x_0)$	$2A$	0
R2	$\gamma_{t-x}^{(1)} + \gamma_{t-x}^{(2)} (x - x_0) + \kappa_t$	$T + 2A - 2$	2
R3	$\gamma_{t-x}^{(1)} + \gamma_{t-x}^{(2)} (x - x_0) + \gamma_{t-x}^{(3)} (x - x_0)^2 + \kappa_t$	$T + 3A - 3$	3

Empirical Results

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Data and Estimation

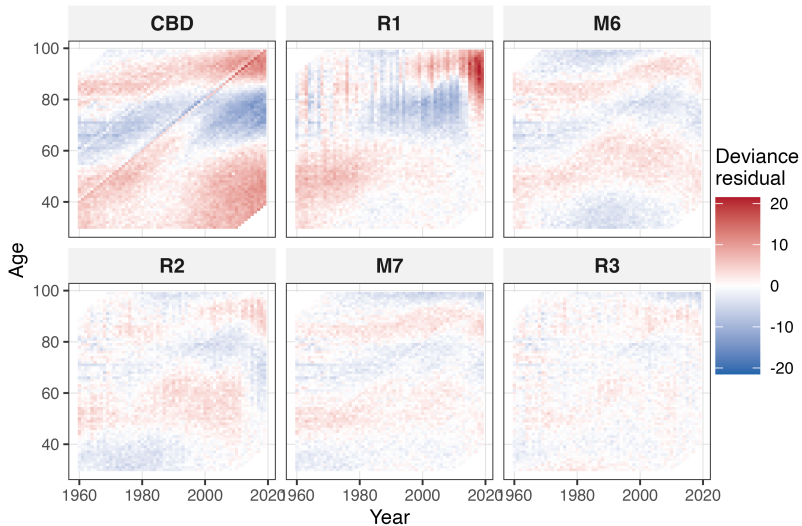
- England and Wales (EW) and United States (US) female mortality from the HMD.
- Ages $x \in [30, 99]$, with $x_0 = 30$.
- Years $t \in [1960, 2019]$.
- Cohorts $c = t - x \in [1861, 1989]$, excluding those with fewer than 10 age-year observations.
- This gives $T = 60$ years and $A = 111$ active cohorts.
- Maximum likelihood estimation with death counts following a Poisson distribution.

Result – England and Wales

Model	<i>n</i>	MAPE		RMSE		Information criteria	
		value	red.	value	red.	Δ AIC	Δ BIC
M5	120	0.11175	—	0.16935	—	—	—
M6	229	0.05208	53.4%	0.07786	54.0%	81.1	80.4
M7	288	0.03568	68.1%	0.05124	69.7%	89.4	88.3
R1	222	0.05795	48.1%	0.07779	54.1%	35.6	34.9
R2	280	0.04675	58.2%	0.07089	58.1%	84.3	83.2
R3	390	0.02839	74.6%	0.04167	75.4%	94.2	92.5

All comparisons are relative to M5; Δ AIC and Δ BIC in thousands, larger is better.

Deviance Residuals – England and Wales

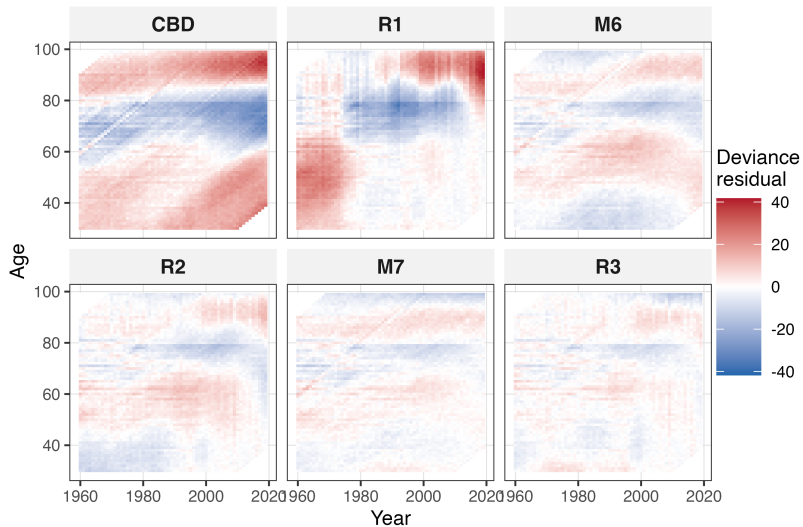


Result – United States

Model	<i>n</i>	MAPE		RMSE		Information criteria	
		value	red.	value	red.	Δ AIC	Δ BIC
M5	120	0.12422	—	0.16123	—	—	—
M6	229	0.05136	58.7%	0.07016	56.5%	610.6	609.9
M7	288	0.03008	75.8%	0.04040	74.9%	676.0	675.0
R1	222	0.07963	35.9%	0.12020	25.4%	184.3	183.7
R2	280	0.04394	64.6%	0.06278	61.1%	635.4	634.4
R3	390	0.02571	79.3%	0.03656	77.3%	692.7	691.0

All comparisons are relative to M5; Δ AIC and Δ BIC in thousands, larger is better.

Deviance Residuals – United States



What the Results Show

- The R3 model ranks first on every criterion, in both countries.
- At every structural level, the redundancy model beats its actuarial counterpart: R1 over M5, R2 over M6, R3 over M7.
- R3 cuts MAPE by **79.3%** (US) and **74.6%** (EW) against CBD, and by **14.5%** and **20.4%** against M7.
- Even a *pure-cohort* model (R1) beats a *pure-period* model (M5): cohort variation in the whole age profile carries information that period effects alone cannot capture.

Discussion

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The Quadratic Term and Health Disparity

Consider $g(\delta) = r\delta$ and $\ln q_x = \text{logit } \delta_x$.

Assume further that

$$\text{logit } \delta_{x_0} \sim N(\mu_\delta, \sigma_\delta^2), \quad r \sim N(\mu_r, \sigma_r^2)$$

with correlation coefficient ρ .

Then

$$\ln \mathbb{E}[q_x] = \left(\mu_\delta + \frac{\sigma_\delta^2}{2} \right) + (\mu_r + \rho\sigma_\delta\sigma_r) \cdot (x - x_0) + \frac{\sigma_r^2}{2} \cdot (x - x_0)^2$$

The curvature comes entirely from *heterogeneity* σ_r^2 in the ageing rate.

- *The quadratic term as a health-disparity index*

$$\gamma_{t-x}^{(3)} \approx \frac{1}{2} \sigma_{r,t-x}^2$$

- *Life-cycle planning with observed frailty*

$$d\delta_x = r \delta_x (1 - \delta_x) dx + \sqrt{\frac{r}{N} \delta_x (1 - \delta_x)} dW_x$$

- *Multi-population modelling, with I indexing sub-populations*

$$\text{logit } \delta_{x_0} \mid I = k \sim N(\mu_{\delta,k}, \sigma_{\delta,k}^2), \quad r \mid I = k \sim N(\mu_{r,k}, \sigma_{r,k}^2)$$

Summary

- Modelling ageing as the loss of biological redundancy makes logit δ_x *exactly* affine in age. The defining structure of the CBD model is therefore derived, not assumed.
- The redundancy parameters belong to an organism and are fixed at birth, so they attach to a *birth cohort* rather than a calendar year.
- This yields the R1–R3 models, which outperform M5–M7 at every structural level, on both US and EW data and on every criterion.
- The quadratic age term gains a meaning it never had: a measure of *health disparity* within the population.

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