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Deprivation as an Earlier Entry onto the Mortality Trajectory: Modelling Life Years Lost from Gompertz–Makeham Age Shift

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Abstract: Background: Deprivation is associated with shorter life expectancy, higher service use and substantial resource demands, particularly in inner-city populations. However, clinical and policy responses often rest on qualitative notions of "accelerated ageing" and do not quantify deprivation as a reproducible structural change in mortality. Aim: To model deprivation as an earlier entry onto the standard mortality trajectory and estimate the resulting life-years lost (LYL) for males and females across adult life. Model: Modelling study using national life-table parameters and Gompertz–Makeham age-shift scenarios. Methods: Age-specific mortality was described by the Gompertz–Makeham law parameterised from Irish Life Table 17 for males and females, providing sex-specific baseline life expectancy from ages 40–85 years. Deprivation was represented as a pure age shift on this trajectory, without altering the Gompertz slope: a 12-year shift for males and a 9-year shift for females. For each starting age, remaining life expectancy and LYL were calculated as the difference between baseline and deprivation life expectancy. To contextualise deprivation, we constructed proportional-hazard reference curves with 2-fold and 4-fold constant excess hazards and compared their LYL profiles with the deprivation curves. Results: Under the male baseline, remaining life expectancy fell from about 35 years at age 40 to 8 years at age 85; a 12-year age shift produced an average LYL penalty of approximately 8 years between ages 40 and 85, declining from 9.4 to 3.4 years. For females, a 9-year age shift yielded an average LYL of about 6 years, with LYL decreasing from 7.6 to 3.8 years over the same age range. In both sexes, the deprivation LYL trajectories lay between the 2-fold and 4-fold hazard benchmarks, consistent with a stable excess hazard close to two-fold across adult life. The implied standardized mortality ratio rose only slightly with age, from 2.04 to 2.20 in males and from 1.60 to 1.80 in females, approaching the theoretical Gompertz limits expected from the calibrated age shifts. Conclusion: Deprivation is modelled as earlier entry onto the mortality trajectory without a need to assume altered acceleration constant of the Gompertz–Makeham equation. LYL trajectories lay somewhat above the 2-fold hazard benchmark, but with a stable and consistent excess hazard across adult life

Keywords: Deprivation, Gompertz-Makeham, law of mortality, life years lost (LYL)

INTRODUCTION

Deprivation, defined by Townsend as a state of "observable and demonstrable disadvantage relative to the local community to which an individual belongs(1), is associated with an increased population mortality (2, 3). UK data has indicated that for males and females from the most deprived areas had a life expectancy 9.0 and 6.9 years shorter than males in the least deprived areas (4). The reasons may include life style choices or the concept of health literacy, where educational levels and acquired skills in empower healthcare decisions promoting good health (5, 6). Our traditional Irish Deprivation Index was devised

by the SAHRU investigators (Trinity College, Dublin), using principle components analysis (PCA), a weighted combination (originally five including overcrowding) of four indicators, relating to unemployment, social class, type of housing tenure and car ownership (7); this methodology is very similar to other classical Deprivation Indexes, including those by Townsend (1) and Carstairs (8). The concept of Deprivation then is one of aggregate community influence rather than at the level of individual personal disadvantage. In any event there is a need to understand the how deprivation influences the health trajectory and ultimately life span. Over the past two decades, policy responses have focused on targeted programmes in high-deprivation areas—enhanced primary care access, case-management, outreach clinics, and various incentive schemes (9). These interventions are well intentioned, but their impact on hard outcomes such as life expectancy has often been modest and difficult to sustain (10). From the clinician's perspective in an inner-city setting, emergency admissions and multimorbidity remain stubbornly concentrated in deprived neighbourhoods despite these efforts.

Mathematical mortality models suggest a reason for this resilience. If deprivation primarily shifts individuals earlier along the same Gompertz–Makeham law of mortality trajectory (11, 12) — advancing the onset of comorbidity and frailty—then the resulting loss of life expectancy is structural, embedded in the age-hazard relationship rather than an episodic excess that can be reversed quickly. In such a framework, deprivation behaves like a fixed "biological age advance" that delivers a nearly constant two-fold excess hazard across adult life, translating into large LYL at 40–50 years but only modest recoverable time at later ages.

In this study we formalise that intuition by modelling deprivation as a sex-specific age shift on the Irish Life Table 17 Gompertz–Makeham baseline and positioning the resulting life-years-lost against Skriver proportional-hazard benchmarks (rates of x2 and x4). Our aim is to quantify where deprivation sits on the multimorbidity risk spectrum for males and females, and to show that much of the associated life-expectancy gap reflects a stable, structurally embedded disadvantage rather than a deficit that can be rapidly eliminated by isolated service interventions.

METHODS

Baseline Life-table Model

We modelled age-specific mortality using the Gompertz-Makeham law of mortality $\mu(t) = c + be^{\wedge}\{kt\}$ where $\mu(t)$ is the mortality hazard at age *t*, *c* is the age-independent Makeham term (violence and accidents), and *b* and *k* are the Gompertz scale and rate parameters governing the exponential increase in mortality with age. Baseline parameters were taken from Irish Life Table 17 (2015–2017) separately for males and female- (males *c* = 0.0007, *b* = 0.000307, *k* = 0.066; analogous values for females), providing sex-specific reference life expectancy from ages 40 to 85 years.

Deprivation Model

Deprivation was conceptualised as an earlier entry onto the same Gompertz-Makeham trajectory, rather than a different ageing process. For each sex we therefore represented

deprivation as a pure age shift of the baseline hazard: $\mu_{\mathrm{dep}}(t) = c + b\exp(k(t + \Delta))$ where Δ is the deprivation age-shift parameter. Based on iterative calibration to yield plausible life-years-lost (LYL) penalties while preserving a stable excess hazard across age, we used Δ =12 years for males and Δ = 9 years for females. No additional steepening of the Gompertz term was introduced (slope multiplier = 1.0 in both sexes). In addition, for each sex we calculated the implied age-specific standardized mortality ratio (SMR) of the deprivation model as the ratio of the shifted Gompertz–Makeham hazard to the baseline hazard at the same chronological age, to quantitate the age-shift framework as a constant proportional hazard across adult life.

Life-years Lost and Skriver Comparators

For each sex we computed remaining life expectancy from ages 40 to 85 under the LT17 baseline and under the deprivation model, and defined LYL as the difference between these two life expectancies at each starting age. To position deprivation on the multimorbidity risk spectrum, we constructed Skriver-style constant proportional hazards of 2-fold (Skriver-x2) and 4-fold (Skriver-x4) relative to LT17 by scaling the baseline hazard and recomputing LYL from ages 40 to 85. The male and female deprivation curves were then compared with proportional reference LYL trajectories, and differences (Δ-Skriver) were tabulated at 5-year age intervals (Tables 1 and 2).

RESULTS

Males: Under the LT17 male baseline, remaining life expectancy declined from approximately 35 years at age 40 to 8 years at age 85 (Table 1). Applying a 12-year Gompertz-Makeham age shift to represent deprivation produced an average life-years-lost (LYL) penalty of about 8 years between ages 40 and 85, with LYL declining from 9.4 years at age 40 to 3.4 years at age 85. The corresponding deprivation LYL trajectory lay consistently between Skriver-x2 and Skriver-x4, exceeding Skriver-x2 by roughly 3-4 years at each age (Δ-Skriver 2.8-3.9 years), and implying an approximately two-fold excess hazard versus LT17 across the adult age range (Figure 1, Table 1).

Table 1: Male deprivation versus Skriver-x2: life-years lost by age 40–85 years

Age	Deprived	Skriver x2	Δ- Skriver
40	9.4	6.6	2.8
45	8.9	5.6	3.3
50	8.4	4.8	3.6
55	7.8	4.0	3.8
60	7.1	3.2	3.9
65	6.4	2.5	3.9
70	5.6	1.8	3.8
75	4.9	1.2	3.7
80	4.1	0.6	3.5
85	3.4	0.1	3.3

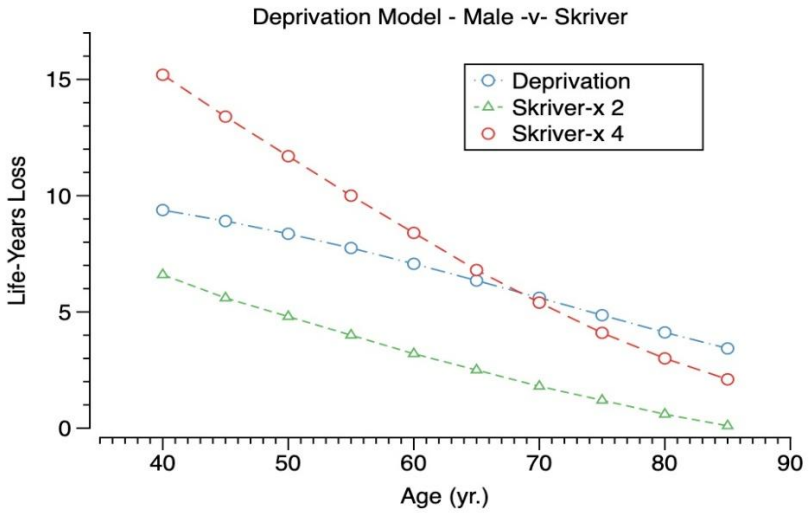

Figure 1: Male life-years-lost trajectories for deprivation and Skriver-based x2 and x4 hazards, ages 40-85

Females: For females, the LT17 baseline projected higher remaining life expectancy at each age, and deprivation was represented by a 9-year Gompertz-Makeham age shift. This yielded an average LYL penalty of about 6 years from ages 40 to 85, with LYL decreasing from 7.6 years at age 40 to 3.8 years at age 85 (Table 2). The female deprivation curve likewise lay between Skriver-x2 and Skriver-x4, with A-Skriver increasing from 1.1 years at age 40 to around 3.0 years at ages 70-75 before narrowing slightly at 85 (2.8 years). Overall, deprivation implied an approximate two-fold excess hazard for females relative to the LT17 baseline, mirroring the male pattern but at lower absolute LYL values (Figure 2, Table 2).

Table 2: Female deprivation versus Skriver-x2: life-years lost by age 40–85 years (with Deprived, Skriver-x2, Δ-Skriver).

Age	Deprived	Skriver x2	Δ- Skriver
40	7.6	6.5	1.1
45	7.4	5.8	1.6
50	7.1	5.1	2.0
55	6.8	4.4	2.4
60	6.4	3.7	2.7
65	6.0	3.1	2.9
70	5.5	2.5	3.0
75	5.0	1.9	3.1
80	4.4	1.4	3.0
85	3.8	1.0	2.8

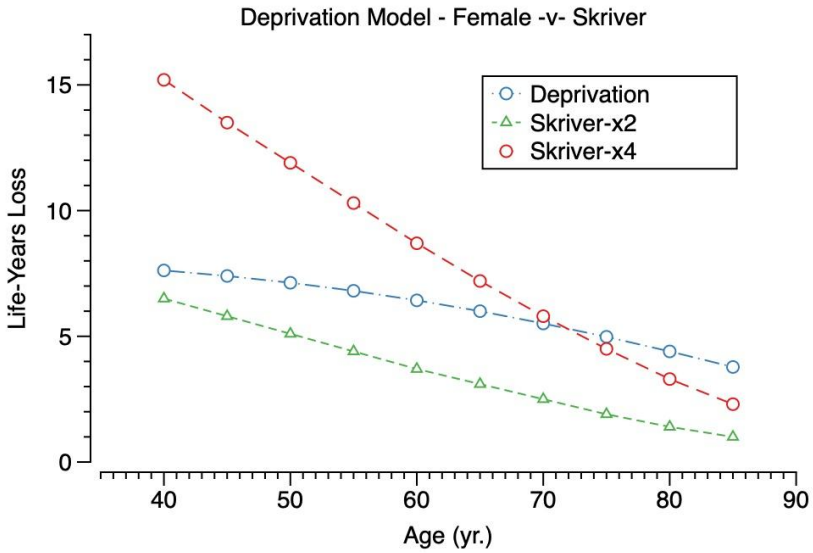

Figure 2: Female life-years-lost trajectories for deprivation and Skriver-based x2 and x4 hazards, ages 40-85

Internal Validation of the Age-Shift model

To verify that the age-shift framework was internally consistent, we tested whether deprived life expectancy calculated by subtracting LYL from the baseline (LE t − LYL) matched baseline life expectancy looked up at the offset age (LE at age+ Δ). For males, model-predicted deprived LE tracked baseline LE at age+12 with near-perfect concordance across all ages (R^2 = 0.9999, maximum difference 0.05 years). For females, model-predicted deprived LE tracked baseline LE at age+9 similarly well (R^2 = 0.999), though with slightly larger differences at older ages (maximum 0.75 years at age 80–85), likely reflecting residual calibration variation in the female Gompertz–Makeham parameters (Figure 3). This validation confirms that the sex-specific age shifts (12 years for males, 9 years for females) reproduce the observed deprivation LYL penalties as a pure horizontal displacement on the mortality trajectory without requiring any change in the Gompertz slope parameter

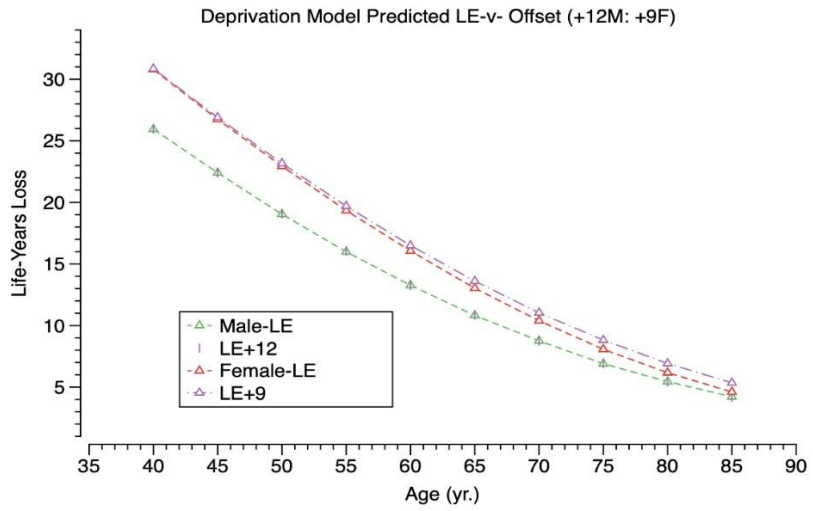

Figure 3: Internal validation of sex-specific age-shift models by offset test. Deprived life expectancy (solid lines with triangle markers: Male-LE in green, Female-LE in red)

versus baseline life expectancy at offset ages (dashed lines: LE+12 for males, LE+9 for females) across ages 40–85 years. Near-perfect tracking in both sexes (males R^2=0.9999; females R^2=0.999) demonstrates that deprivation operates as a horizontal age displacement of 12 years (males) and 9 years (females) without altering the exponential mortality slope

Mathematical Equivalence to SMR

To further characterise the age-shift model, we calculated the implied standardized mortality ratio (SMR) at each age as the ratio of the deprivation hazard to the baseline hazard. For males, the 12-year shift yielded an SMR increasing from 2.04 at age 40 to 2.20 at age 85 (mean 2.15); for females, the 9-year shift yielded an SMR increasing from 1.60 to 1.80 (mean 1.73) (Figure 4). These values approached the theoretical Gompertz limits of $e^{k\Delta}$, namely 2.21 for males and 1.81 for females, confirming that the deprivation shift behaves as an approximately constant proportional hazard across adult life, with slight age-related increase due to the Makeham term.

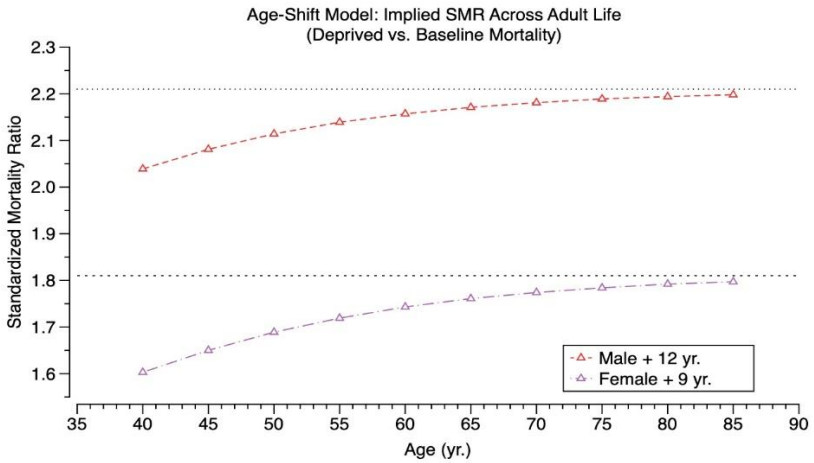

Figure 4: Implied standardized mortality ratio (SMR) of the deprivation age-shift model across ages 40–85 years. The slight age-related increase reflects the Gompertz-Makeham structure, in which the age-independent Makeham term has greater relative influence at younger ages and progressively less influence as exponential senescence dominates

DISCUSSION

The principal finding of this study is that Deprivation can be represented parsimoniously as an earlier entry onto the same adult mortality trajectory, rather than as a fundamentally different mortality process. Applying a sex-specific horizontal shift to the Gompertz–Makeham (11, 12) baseline generated plausible life-years-lost penalties across ages 40–85 years, averaging about 8 years in males and 6 years in females, and positioned Deprivation between the 2-fold and 4-fold benchmark hazard curves. The implication is that Deprivation applies a "constant lead-time shift" rather than an accelerating biological breakdown – its impact appears greatest however in mid-life (40 – 65-yr.) where the gap between Deprivation and the life table is widest.

In strict Gompertz–Makeham terms, "accelerated ageing" would usually imply a steeper slope, that is, a larger Gompertz rate parameter *k*, with mortality rising faster with each additional year of age (11, 12). That is not what was this modelled revealed. Instead, as slope was unchanged, Deprivation is due to a horizontal age shift of 'entry point' onto the mortality curve. The present model is consistent with earlier biological or actuarial ageing rather than constant accelerated ageing over time. Internal validation by the offset test confirmed that this interpretation is mathematically coherent: the model-predicted deprived life expectancy matched baseline life expectancy at the offset age (Δ = 12 years for males, 9 years for females) with $R^2 > 0.999$ in both sexes (Figure 3).

A further mathematical insight is that the age-shift model is closely related to a proportional-hazard interpretation. In a pure Gompertz model, a fixed age shift of Δ years is exactly equivalent to a constant hazard multiplier of $e^{k\Delta}$. In the present Gompertz–Makeham framework, the same relationship holds approximately rather than exactly, because the age-independent Makeham term dilutes the ratio at younger ages; consequently, the implied SMR rises slightly with age before converging toward the Gompertz limit (Figure 4). Calculating without the Makeham term eliminates this apparent discrepancy. This is important conceptually, because it shows that the deprivation model is not only an intuitive "earlier entry" formulation, but also mathematically compatible with the epidemiological concept of excess relative risk.

Viewed from this perspective, the results provide a numerical interpretation of what clinicians often observe in actual clinical practice. Deprived populations generate disproportionate emergency admissions (13), carry higher burdens of multimorbidity (1, 3), and appear older than their chronological age would suggest. The age-shift model translates that observation into numbers: approximately a 12-year effective age advance in males and a 9-year in females. This does not explain the mechanism of Deprivation induced-ageing, but it provides a semi-quantitative method of calibration.

The wider literature is well-known. Classical deprivation research describes substantial mortality differences between more and less deprived populations (1, 14); more recent work reveals that Deprivation is associated with earlier onset of multimorbidity (13), greater healthcare use (15), and worse outcomes across the life course (10, 16). Studies of frailty trajectories similarly suggest that deprived groups enter later life with higher levels of deficit accumulation, which is compatible with the idea that much of the mortality penalty is already established by mid-life (1, 3, 17, 18).

This model suggests the as deprivation reflects a structurally embedded shift in where a population sits on the mortality curve, then later interventions may improve access, experience, and possibly short-term utilisation, yet still have limited capacity to restore already embedded LYL. The model therefore ought not to focus improvement initiatives based on assumptions of 'accelerated ageing' ongoing over time rather than accumulated over a specific life period. At the same time, it does not imply therapeutic nihilism. Rather, it suggests that interventions capable of influencing life expectancy meaningfully are more likely to need to begin at the appropriate time, when risk exposure is operating at greatest effect, whereas later-life interventions may be more anticipated to more improve by quality of life, functional preservation, or healthcare efficiency than by major recovery of lost survival time.

The question as to when the age shift actually occurs cannot of course be answered from a model that simply states that, between ages 40 and 85, deprived individuals behave as though they are already older on the mortality trajectory. However, life course epidemiology (19) may envisage trajectories by which shifts may occur at different time point as in foetal life (20), or the critical teenage / early adult years; cumulative risk may accumulate due for various exposures or earlier onset of chronic disease (21). On that interpretation, the Δ-shift used here should be viewed not as a summary parameter of the cumulative impact of long-term disadvantage.

Future work should test whether similar age-shift values are observed in other national life tables and deprivation indices, and whether the shift can be estimated directly from linked longitudinal data. Whether small changed in slope might improves model fit in some settings could be considered; were the slope to remain constant, the deprivation penalty could be quantitatively in multimorbidity risk strata (such as the ERFC cardiometabolic benchmarks) to position social disadvantage on the same numerical spectrum as that for chronic disease. More broadly, the approach could be extended by integrating deprivation with specific multimorbidity datasets so that social disadvantage and disease burden can be expressed within a common life-years-lost framework. Additionally, longitudinal biomarker data tracking individuals from adolescence through mid-life could help identify the timing and rate of deprivation-related age-shift accumulation, testing whether the shift is established early (before age 40) or accumulates progressively across the life course.

Limitations: Several limitations follow directly from the simplicity of the model. First, it is a mathematical model of modification of Gompertz-Makeham parameters to emulate the observed mortality loss and as such has not been tested over observed longitudinal cohorts. Second, the model revelations that deprivation changes position on the mortality curve but not the slope of the Gompertz component has not been tested. It is however, mathematically sound and internally consistent. Third, the framework uses Irish Life Table 17 as the baseline and should be interpreted relative to that reference population. Having said that a life table is a life table. Finally, deprivation is an area-level construct that cannot fully capture individual-level resilience, disease mix, or behavioural heterogeneity.

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