

Review of: "Computer Analysis of Stochastic Aging According to the Gompertz-Makeham Mortality Law"

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The paper introduces a stochastic approach to the Gompertz-Makeham mortality law by modeling its parameters A, B, and C as Gaussian random variables. The author derives analytical expressions for the first four central probabilistic moments of the mortality rate and conducts numerical experiments using the MAPLE software. While integrating stochastic elements into mortality modeling is conceptually valuable, several significant concerns arise regarding the theoretical assumptions and practical applicability of the model.

A primary issue lies in the treatment of the parameters A, B, and C as Gaussian random variables. In the traditional Gompertz-Makeham mortality model, these parameters are non-negative due to their biological and demographic interpretations. Parameter A represents the baseline mortality rate from age-independent risks, which cannot be negative. Parameter B characterizes the exponential increase in mortality with age, reflecting senescence; it must be non-negative because mortality generally increases with age in biological populations. Parameter C accounts for age-independent constant hazards such as environmental risks, which are inherently non-negative.

By modeling these parameters as Gaussian random variables, negative values become possible, violating the fundamental assumption that mortality rates cannot be negative. Negative values for A, B, or C lack biological meaning and could lead to mathematically anomalous behaviors, such as negative mortality rates (when A or C are negative), undefined probabilistic moments (when B is negative), or identifiability problems (when B is zero). This undermines the validity of the model and may result in unrealistic or misleading calculations. This concern is supported by the foundational works on the Gompertz-Makeham model, which emphasize the necessity of non-negative parameters (**Gompertz, 1825; Makeham, 1860**).

Another concern is the assumption that A, B, and C are uncorrelated random variables. Empirical research in demography has shown that these parameters are often correlated. For instance, populations with higher baseline mortality (A) often face greater extrinsic risks (C), influenced by shared environmental and genetic factors. **Vaupel et al. (1979)** demonstrated that individual frailty and heterogeneity significantly impact mortality dynamics, reinforcing that parameters like A and C are interrelated due to shared environmental and genetic factors. Ignoring these correlations oversimplifies the complexity of mortality dynamics and fails to capture key interdependencies essential for understanding how populations behave under shared risk environments.

The assumption of independence may lead to underestimation or mischaracterization of variability in mortality rates,

particularly in aging populations where variability increases with age. This oversimplification could affect the accuracy of the derived probabilistic moments and numerical simulations, making the model less reliable. **Wilmoth and Horiuchi (1999)** discussed the importance of considering parameter correlations in mortality models to accurately reflect demographic realities.

An additional issue pertains to the identifiability of the model when C is treated as a random variable. Identifiability refers to the ability to uniquely estimate model parameters from the available data. When C is random, it may not be possible to distinguish the effects of C from those of other parameters or random components in the model. This problem is recognized in models like the gamma-Gompertz, where randomness is not linked to C precisely because of identifiability concerns (**Missov & Vaupel, 2015**). Introducing randomness into C without addressing these issues may lead to a model where the parameters cannot be reliably estimated or interpreted, reducing the model's practical usefulness.

The model also does not account for mortality deceleration at advanced ages—a well-documented phenomenon where the rate of increase in mortality slows down in older age groups. Studies such as Horiuchi and **Coale (1990)** and **Vaupel et al. (1998)** have documented mortality deceleration and its implications for mortality modeling. The traditional Gompertz-Makeham model is known to deviate at advanced ages due to this deceleration. By not addressing this, the model's applicability to late-life dynamics is limited, and it may not accurately reflect the mortality patterns observed in real populations.

While the mathematical derivations and computational implementations are rigorous, the paper lacks empirical grounding. There is no validation of the model against actual mortality data from human or animal populations. Without empirical validation, theoretical predictions—such as the dominance of parameter B in influencing mortality rates—remain unsubstantiated. This limits the model's relevance to demographic research and its usefulness in practical applications. The importance of empirical validation in demographic models is emphasized in works like **Preston et al. (2001)**.

Furthermore, the paper does not incorporate insights from seminal works in demographic modeling, such as James W. Vaupel's study on the impact of heterogeneity in individual frailty on mortality dynamics (**Vaupel, Manton, & Stallard, 1979**). Vaupel emphasized the significance of individual variability and frailty in shaping population-level mortality trends. Incorporating frailty into the stochastic framework could enhance the model's realism and alignment with empirical observations.

Despite these concerns, the paper has notable strengths. Introducing stochastic elements into the Gompertz-Makeham model enriches our understanding of variability in mortality rates. The analytical derivation of probabilistic moments adds depth to the mathematical study of stochastic aging. The use of symbolic algebra and numerical implementation using MAPLE demonstrates methodological proficiency and offers precise exploration of stochastic aging dynamics. The paper also opens avenues for extending the model to include other probabilistic methods, distributions, and more advanced modeling of mortality rates as stochastic processes.

In conclusion, while the paper makes a meaningful contribution to the mathematical study of stochastic aging, the theoretical assumptions limit its robustness and practical applicability. To enhance the model's relevance and accuracy, it

is recommended to modify the model to enforce non-negative values for A , B , and C , perhaps by using probability distributions that are bounded below by zero, such as log-normal or gamma distributions. Accounting for the correlations between parameters, particularly between A and C , would reflect the interdependencies observed in empirical data. Addressing the identifiability issues associated with making C random is crucial; perhaps randomness should be introduced in a way that does not compromise parameter estimation, as seen in models like the gamma-Gompertz where randomness is linked to other components. Extending the model to capture mortality deceleration at advanced ages would improve its applicability to late-life mortality dynamics. Comparing model predictions with actual mortality data would substantiate theoretical findings and adjust the model accordingly. Incorporating concepts from frailty models to account for individual variability could enhance the model's biological plausibility.

By addressing these concerns, the paper could significantly advance the understanding of stochastic mortality dynamics and provide a more accurate and practical tool for demographic research.

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