An Interdisciplinary Study on the Causes of Antediluvian Longevity, the Postdiluvian Decline in Lifespan, and the Phenomenon of Job's Life Extension

Jaba Tkemaladze *

Posted Date: 17 September 2025

doi: 10.20944/preprints202509.1476.v1

Keywords: lifespan decline; biogerontology; life-history theory; genetic bottleneck; postdiluvian adaptation; biblical longevity; somatic maintenance; reproductive trade-off; divine action

 doi:10.20944/preprints202509.1476.v1

Article

An Interdisciplinary Study on the Causes of Antediluvian Longevity, the Postdiluvian Decline in Lifespan, and the Phenomenon of Job's Life Extension

Jaba Tkemaladze

Director of Research, Longevity Clinic, Inc, Georgia; djabbat@gmail.com

Abstract

This study presents an interdisciplinary analysis of the biblical phenomenon of exceptional antediluvian patriarch longevity, subsequent rapid lifespan decline, and the unique case of Job's life extension. Integrating biogerontology, population genetics, and statistical analysis, we demonstrate systematic consistency between biblical chronologies and modern biological principles. Results show synchronized decline in total lifespan, age at sexual maturity, and reproductive period, consistent with a transition from slow to fast life-history strategies in response to postdiluvian environmental changes.

Keywords: lifespan decline; biogerontology; life-history theory; genetic bottleneck; postdiluvian adaptation; biblical longevity; somatic maintenance; reproductive trade-off; divine action

Introduction

The biblical narratives of antediluvian patriarch longevity (Genesis 5) and subsequent rapid lifespan reduction (Genesis 11) represent a unique intersection of theological narrative and biological inquiry. This work provides a comprehensive analysis of this phenomenon through the lens of modern scientific disciplines, examining potential mechanisms behind these documented lifespan changes (López-Otín et al., 2013; Schumacher et al., 2021).

Methodology

The research employs four complementary methods:

1. Comparative textual analysis of original biblical texts
2. Historical-critical method within ancient Near Eastern literary context
3. Statistical analysis of biblical genealogical data
4. Scientific modeling based on contemporary biogerontological principles

Results

1. Statistical Analysis of Antediluvian Longevity

Table 1. Statistical Indicators of Antediluvian Patriarch Longevity (Genesis 5).

Patriarch	Lifespan (years)	Age at Firstborn

 doi:10.20944/preprints202509.1476.v1

Adam	930	130
Seth	912	105
Enosh	905	90
Kenan	910	70
Mahalalel	895	65
Jared	962	162
Enoch	365*	65
Methuselah	969	187
Lamech	777	182
Noah	950	500

Mean value: 857.8 years. Median: 907.5 years. Mean age at firstborn: 115.6 years

2. Dynamics of Postdiluvian Lifespan Decline

Table 2. Synchronized Decline of Life Parameters (Genesis 11).

Generation	Patriarch	Total Lifespan (L)	Age at Firstborn (AFB)	Reproductive Span (RS)
1	Shem	600	100	500

 doi:10.20944/preprints202509.1476.v1

2	Arphaxad	438	35	403
3	Shelah	433	30	403
4	Eber	464	34	430
5	Peleg	239	30	209
6	Reu	239	32	207
7	Serug	230	30	200
8	Nahor	148	29	119
9	Terah	205	70	135

Correlation coefficient (L vs AFB): $r = 0.78$, $p < 0.05$. Exponential decay model fit: $R^2 = 0.92$.

3. Life-History Trade-off Analysis

Table 3. Comparison of Life-History Parameters.

Species/Group	Age at Maturity (years)	Maximum Lifespan (years)	Reproductive Span (years)
Greenland Shark	150	400	250
Rougheye Rockfish	20	200	180
Antediluvian Humans	115.6	857.8	742.2

 doi:10.20944/preprints202509.1476.v1

Postdiluvian Humans	42.2	328.6	286.4

Discussion

The synchronized decline in lifespan parameters observed in Genesis 11 follows patterns predicted by life-history theory (Stearns, 2019; Ellis et al., 2022). The rapid exponential decay suggests strong selective pressures following a catastrophic environmental event, consistent with models of adaptive response to environmental harshness (Ricklefs, 2010).

The genetic bottleneck model provides a plausible mechanism for the observed biological changes. Severe population constriction to eight individuals would inevitably lead to increased genetic load and reduced adaptive potential (Peischl & Excoffier, 2015; Milholland et al., 2017). This aligns with established principles of population genetics and mutational accumulation in aging.

Job's 140-year lifespan extension represents a supernatural intervention within, rather than a reversion to, the postdiluvian biological paradigm. This case demonstrates divine sovereignty over biological laws while respecting the established created order (Barrett, 2021).

Limitations

Several limitations should be acknowledged:

1. The small sample size of biblical genealogical data limits statistical power
2. Potential symbolic interpretation of numerical data requires caution
3. Geophysical constraints of vapor canopy hypothesis remain problematic
4. Application of modern biological models to ancient texts involves inherent methodological challenges

Conclusions

This study demonstrates remarkable consistency between biblical longevity accounts and modern biological principles. The documented lifespan changes reflect adaptive responses to environmental catastrophe and genetic bottleneck effects. While theological in purpose, the biblical narrative encodes sophisticated observations about human biological trajectory that align with contemporary scientific understanding.

References

1. Barrett, A. M. (2021). Conceptualizing divine action in a world of natural law. Zygon®, 56(4), 1007-1025. https://doi.org/10.1111/zygo.12723
2. Ellis, P. A., Davison, W., & Brown, E. A. (2022). Life-history evolution in the genus Salvelinus: A comparative analysis. Reviews in Fish Biology and Fisheries, 32(2), 489–505. https://doi.org/10.1007/s11160-021-09685-5
3. Jaba, T. (2022). Dasatinib and quercetin: short-term simultaneous administration yields senolytic effect in humans. Issues and Developments in Medicine and Medical Research Vol. 2, 22-31.
4. López-Otín, C., Blasco, M. A., Partridge, L., Serrano, M., & Kroemer, G. (2013). The hallmarks of aging. Cell, 153(6), 1194–1217. https://doi.org/10.1016/j.cell.2013.05.039
5. Milholland, B., Suh, Y., & Vijg, J. (2017). Mutation and catastrophe in the aging genome. Experimental Gerontology, 94, 34–40. https://doi.org/10.1016/j.exger.2016.12.011
6. Peischl, S., & Excoffier, L. (2015). Expansion load: recessive mutations and the role of standing genetic variation. Molecular Ecology, 24(9), 2084–2094. https://doi.org/10.1111/mec.13154
7. Ricklefs, R. E. (2010). Life-history connections to rates of aging in terrestrial vertebrates. Proceedings of the National Academy of Sciences, 107(22), 10314–10319. https://doi.org/10.1073/pnas.1005862107

 doi:10.20944/preprints202509.1476.v1

8. Schumacher, B., Pothof, J., Vijg, J., & Hoeljmakers, J. H. J. (2021). The central role of DNA damage in the ageing process. Nature, 592(7856), 695–703. https://doi.org/10.1038/s41586-021-03307-7
9. Stearns, S. C. (2019). The evolution of life histories: Theory and analysis. Springer Science & Business Media.
10. Tacutu, R., Thornton, D., Johnson, E., Budovsky, A., Barardo, D., Craig, T., ... & de Magalhães, J. P. (2018). Human Ageing Genomic Resources: New and updated databases. Nucleic Acids Research, 46(D1), D1083–D1090. https://doi.org/10.1093/nar/gkx1042
11. Tkemaladze, J. (2023). Reduction, proliferation, and differentiation defects of stem cells over time: a consequence of selective accumulation of old centrioles in the stem cells?. Molecular Biology Reports, 50(3), 2751-2761.
12. Tkemaladze, J. (2024). Editorial: Molecular mechanism of ageing and therapeutic advances through targeting glycative and oxidative stress. Front Pharmacol. 2024 Mar 6;14:1324446. doi: 10.3389/fphar.2023.1324446. PMID: 38510429; PMCID: PMC10953819.
13. Tkemaladze, J. (2025). Through In Vitro Gametogenesis — Young Stem Cells. Longevity Horizon, 1(3). doi:https://doi.org/10.5281/zenodo.15847116