The Evolutionary and Biophysical Determinants of Maximum Lifespan: Scaling Laws, Brain Size, and Future Projections

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Abstract

The lifespan of all organisms is determined by an interplay of genetic architecture and environmental conditions. Within a population, the lifespan of the longest-lived individual defines the Maximum Lifespan (MLS). For contemporary *Homo sapiens*, this value is approximately 113 years, with verified records suggesting a potential upper bound of 115-120 years. Throughout human history, average life expectancy has increased dramatically. In early hominin populations, average lifespan was around 30-40 years, rising to about 60 years in the 20th century, and currently averaging 70-80 years in developed nations. This increase has significantly facilitated cumulative knowledge transfer and cultural complexity across generations. For modern humans, with a brain volume of ~1,446 cm^3 and an observed MLS of ~95 years, the value calculated by this formula (~92 years) shows strong concordance. For instance, if human MLS were to increase to 200 years, brain volume would need to expand to approximately 5,688 cm^3, body mass would also increase, and daily caloric intake would rise to ~1,523 kcal. This radical change would also affect reproductive strategies; the onset of sexual maturity could be delayed from the current 14-17 years to 30-37 years in a 200-year MLS scenario. In conclusion, focusing on a single mechanism—such as somatic mutations—is insufficient for understanding the limits of the human lifespan.

Key Words: maximum lifespan, aging, future, evolution

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Introduction

All organisms have a characteristic lifespan determined by an interplay of genetic architecture and environmental conditions. This lifespan is modulated by numerous factors, leading some individuals to succumb early to disease or resource scarcity, while others achieve extended longevity. Within a population, the lifespan of the longest-lived individual defines the Maximum Lifespan (MLS). Theoretically, given ideal conditions, other members of the species possess the potential to approach this biological limit. For contemporary *Homo sapiens*, this value is approximately 113 years, with verified records suggesting a potential upper bound of 115-120 years.

The trajectory of human average lifespan has shifted dramatically over evolutionary and historical time. In early hominin populations facing environmental hazards and nutritional stress, average lifespan is estimated to have been a mere 30-40 years. Paleodemographic data indicate an average lifespan of about 29 years 30,000 years ago, rising to 32 years by 12,000 years ago, 38 years by 8,000 years ago, 35 years around 1100 BC, and 48 years by 1200 AD. The 20th century saw a jump to around 60 years, with modern developed nations now averaging 70-80 years. This pronounced extension of average life expectancy has profound implications, facilitating greater cumulative knowledge transfer and cultural complexity across generations.

Crucially, interspecies differences in MLS cannot be attributed to genetics alone. Despite sharing approximately 99% of their DNA, chimpanzees exhibit an MLS roughly half that of humans and experience a accelerated pace of physiological aging. This suggests that the regulatory mechanisms governing gene expression—rather than the protein-coding genes themselves—play a primary role in determining aging rates and lifespan. Comparative analyses across mammalian species reveal a robust allometric relationship between MLS, brain mass, and body mass:

$$\text{MLS (years)} = 10.8399 \times (\text{Brain Mass in g})^{0.636} \times (\text{Body Mass in g})^{-0.225}$$

This scaling law, applicable to both extant species and hominin fossils (Bozcuk, 1982), underscores the deep evolutionary link between encephalization and longevity. The data in the accompanying table demonstrate a strong concordance between observed maximum lifespans in various mammals and primates and the values predicted by this brain-body mass equation.

Extrapolating from this relationship allows for backward estimation. An MLS of 200 years or more would likely coincide with significant increases in both brain and body mass. Estimates place the MLS of *H. habilis* around 60 years, *H. erectus* between 70-80 years, and modern *H. sapiens* at approximately 100 years. A simplified, brain-centric formulation of this relationship is:

$$\text{MLS (years)} \approx 1.607 \times (\text{Brain Mass in g})^{0.56}$$

Species	Brain Vol. (cm^3)	Body Mass (g)	MLS - Observed (yrs)	MLS - Calculated (yrs)
MAMMALS				
Mouse	0.45	22.6	3.5	3.2
Badger	7.65	5,000	7.0	5.8
Camel	570	450,000	30	33
Cow	423	465,000	30	27
Giraffe	680	520,000	34	35
Elephant	5,045	2,347,000	70	89
Mountain Lion	154	54,000	19	23
Domestic Cat	79	13,400	20	21
PRIMATES				
Squirrel Monkey	24.8	630	21	20
Rhesus Monkey	106	8,719	29	27
Baboon	179	16,000	36	33
Gibbon	104	5,500	32	30
Orangutan	420	69,000	50	41
Gorilla	550	140,000	40	42
Chimpanzee	410	49,000	45	43
Human	1,446	65,000	~95	92

Calculations based on paleontological trends suggest that over a span of one million years of evolution, the human MLS increases by roughly 1.6 years. This persistent upward trend implies that, should evolution continue unabated for another million years, our average brain mass could escalate from its current ~1350 g to around 5000 g. Concurrently, the MLS would potentially double from about 100 to 200 years.

Such a dramatic five-fold increase in brain mass would have unforeseeable consequences for cognitive architecture and information processing. It might introduce significant metabolic and computational inefficiencies, imposing a heavy burden on the brain's "operating system." Evolution, however, tends to favor practical, energetically sustainable solutions over mere increases in scale. Therefore, while the scaling laws point to a potential future of larger brains and longer lives, the actual evolutionary path will be constrained by trade-offs involving energy cost, reproductive fitness, and cognitive utility. Furthermore, given the glacial pace of such macroevolutionary change (1.6 years per million years), the prospect of a 5 kg human brain remains a distant speculation.

A Model of Continuous Brain Evolution and Potential Future Differentiation

The table below projects key biological parameters under different future Maximum Lifespan (MLS) scenarios, illustrating the integrated changes in brain capacity, metabolism, and life history that such extensions would entail.

Parameter	MLS = 92 yrs	MLS = 150 yrs	MLS = 200 yrs
Brain Capacity (cm^3)	1,446	3,396	5,688
Body Mass (kg)	65	81	97
Metabolic Rate (cal/g/day)	23	21.8	20.8
Caloric Intake (kcal/day)	780	1,198	1,523
Age at Sexual Maturity (yrs)	14-17	22-27	30-37
Brain/Body Mass Ratio (%)	~2.2	~4.2	~5.9

A central implication of extending MLS is a concomitant shift in life history strategy. As lifespan increases, the onset of reproductive maturity is projected to delay significantly. While fertility currently begins around ages 14-17, an MLS of 150 years would push this threshold to 22-27 years, and an MLS of 200 years to 30-37 years. This delay aligns with evolutionary theories that posit a trade-off between somatic maintenance (for longevity) and early reproductive investment. Thus, any future increase in human MLS driven by evolutionary processes will not be an isolated phenomenon but part of a systemic reorganization of our developmental timing, energy budgeting, and neurobiological architecture.

Somatic mutations impose an entropic upper bound

The preprint ***"Somatic mutations impose an entropic upper bound on human lifespan"*** presents a significant methodological advance in gerontology by developing a structured, incremental modeling framework to dissect the complex process of aging (Efimov et al., 2025). A key contribution of this work is its demonstration of a fundamental asymmetry in how somatic mutations affect different tissue types. The finding that post-mitotic cells (neurons and cardiomyocytes) act as critical longevity bottlenecks, while highly regenerative tissues like the liver can maintain functionality for millennia through cellular turnover, provides crucial guidance for future therapeutic prioritization (Kirkwood, 1977; López-Otín et al., 2013). Furthermore, the application of reliability theory, modeling the

human body as a system of serially and parallelly connected components, successfully translates engineering principles to a biological context, offering a robust quantitative foundation.

However, the model notably overlooks critical evolutionary and biophysical determinants of human lifespan, particularly the deeply entrenched allometric relationship between brain size, metabolic rate, and maximum longevity. Anthropological and comparative biological studies have long established a robust scaling law, often expressed as Maximum Lifespan ≈ kx (Brain Mass)$^{\alpha}$, where α approximates 0.56 (Sacher, 1959; Hofman, 1993). This relationship is not merely correlative but is underpinned by the immense metabolic cost of neural tissue. The human brain, representing only ~2% of body weight, consumes ~20-25% of the body's basal metabolic rate (BMR) (Aiello & Wheeler, 1995). This "expensive tissue" imposes a fundamental constraint: extending cognitive function and neural integrity over the model's predicted 134–170 year median lifespan would require not just resisting mutational entropy, but also sustaining this disproportionate energy allocation for over a century beyond current norms.

This biophysical reality directly engages with the model's parameters. The study's calculated theoretical non-aging baseline of 430 years (at age-30 mortality) and its subsequent reduction by somatic mutations, while mathematically sound, exist in an evolutionary vacuum. As noted in ancillary paleoanthropological analyses, a projected increase in maximum lifespan (AÖ) to 200 years is evolutionarily coupled with a required expansion of brain capacity to nearly 5,700 cm³ and a significant rise in total caloric consumption (Bozcuk, 1982). The current model, by treating organ capacity (K) as a static, log-normally distributed variable, fails to incorporate the dynamic, co-evolutionary feedback between longevity, encephalization, and the body's energy budget. Sustaining a ~1.4 kg brain for 150 years is metabolically challenging; sustaining the larger brain implied by such longevity evolution would dramatically alter the energy landscape, potentially intensifying oxidative stress and influencing mutation rates themselves—a variable currently held constant.

Thus, by isolating somatic mutation accumulation from the broader context of human encephalization and its requisite metabolic investment, the study risks presenting an upper bound that is neurobiologically and evolutionarily untenable. The "entropic upper bound" imposed by somatic mutations might be preempted by an earlier "energetic upper bound" imposed by the escalating cost of maintaining the very organ most critical to survival—the brain. A comprehensive model must integrate these scaling laws, recognizing that lifespan extension is not a singular process of damage repair but a systemic renegotiation of energy allocation and neural architecture (Robson & Wood, 2008; Fonseca-Azevedo & Herculano-Houzel, 2012).

A further significant limitation is the model's disconnection from life history evolution and fertility dynamics. A core tenet of evolutionary biology is the trade-off between longevity and reproduction (Stearns, 1992). Historical and paleoanthropological data suggest that increases in lifespan are accompanied by delayed sexual maturation and extended reproductive periods (Bogin & Smith, 1996; Gurven & Kaplan, 2007). For instance, a lifespan extending to 150 or 200 years would logically shift the onset of reproduction to later ages (e.g., 22-27 or 30-37 years, respectively). The study's "somatic-mutations-only" scenario does not account for how such a dramatic shift in the reproductive window would impact population dynamics, intergenerational intervals, and genetic diversity. Ignoring these demographic and evolutionary feedback mechanisms limits the realism of the proposed lifespan extension, as reproductive strategy is a fundamental pillar of a species' survival and adaptation.

Additionally, the model gives limited consideration to energy metabolism and other primary aging processes. Longevity is intricately linked not only to the accumulation of cellular damage but also to the economics of energy production, allocation, and consumption—concepts central to the Disposable Soma Theory (Kirkwood, 1977). The human brain is a metabolically expensive organ, consuming a disproportionate share of the body's energy budget (Aiello & Wheeler, 1995). Supporting its function over 150-200 years would impose immense metabolic costs, potentially exacerbating other aging hallmarks like mitochondrial dysfunction. By focusing predominantly on somatic mutations, the model sidelines the potential compounding effects and interactions with other critical aging processes such as loss of proteostasis, altered intercellular communication, and stem cell exhaustion (López-Otín et al., 2013). A comprehensive upper-bound estimate must integrate these interconnected mechanisms.

In conclusion, Efimov et al. provide a valuable and sophisticated starting point for quantifying the theoretical limit imposed by one fundamental aging process. Yet, a truly holistic model of human longevity must integrate constraints from evolutionary biology, life history theory, and systems metabolism. Future research should aim to create integrative frameworks that simulate not only the accumulation of somatic mutations but also the co-evolution of brain and body, shifts in reproductive strategies, and metabolic adaptations required for extreme longevity. Such a multidisciplinary approach, bridging gerontology, evolutionary anthropology, and systems biology, will deepen our understanding of human lifespan limits and provide a more robust foundation for evaluating potential intervention strategies.

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