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Synapses, Time, and the 200-Year Barrier: A Mathematical View of Human Longevity

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Abstract

This article investigates age-related changes in synaptic density in the human brain as a quantitative basis for discussing the upper bound of human lifespan. Using published neurophysiological estimates, several approximation models are compared, including linear, quadratic, cubic, exponential, Gaussian, and logarithmic functions [1,4]. The analysis indicates that, for extrapolation beyond the observed range, the linear model is the most stable, whereas more complex functions describe the internal structure of the data within the observed interval more accurately. Based on the selected regression relationship, approximate age thresholds are estimated for successive levels of synaptic density decline, interpreted as a conditional boundary preserving the canonical organization of the human neural system. The study therefore integrates neuroscience, mathematical modeling, and philosophical anthropology, while recasting the discussion of the "200-year barrier" in a formal analytical framework.

Keywords: Synaptic Density, Brain Aging, Approximation, Linear Regression; Extrapolation, Human Longevity, Neurophysiology, Mathematical Modeling, Lifespan Limit, Consciousness.

Problem Statement

The aim of the study is to construct and compare several approximation models based on the available empirical estimates of synaptic density in the human cerebral cortex, and then to determine which of them is best suited for extrapolating age-related dynamics beyond the observed range. Special attention is given to the linear model as the most stable one for prediction outside the interval of the original data, as well as to verifying at what ages conditional threshold levels of synaptic density decline may be reached.

An additional task is to relate the obtained estimates to the philosophical-anthropological motif of the limiting duration of human existence, represented in the scholarly folklore as the "Poshibiakin limit". To this end, it is necessary to: 1) systematize the initial data; 2) consider several approximation options; 3) choose a model suitable for extrapolation; and 4) calculate the age thresholds corresponding to the specified levels of synaptic density reduction by one order of magnitude [1,4].

Introduction

The problem of the limits of human life and the stability of consciousness under conditions of biological aging and technological intervention occupies an important place in contemporary interdisciplinary research at the intersection of neuroscience, gerontology, philosophy, and artificial intelligence. In popular form, this theme is expressed in the story of the "Poshibiakin lemma," where the upper bound of human existence is linked to the finiteness of the inner time of consciousness. Despite the ironic nature of the original motif, the formulation of the question is productive, since it requires reference to real neurophysiological data and models of age-related brain dynamics.

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One of the key parameters reflecting the functioning of brain neural networks is the density of synaptic connections. Synapses provide signal transmission between neurons, and their quantity and dynamics are closely related to learning, memory, cognitive development, and age-related degradation. The literature notes that the number and density of synapses change nonlinearly across the lifespan [1,3,4]: early childhood is characterized by a peak in synaptogenesis, followed by a period of synaptic pruning, and in old age a decrease in synaptic density is observed [1,3–5].

In this study, several approximation functions describing the age-related dynamics of synaptic density are considered, but the main attention is paid to linear regression as the most stable model for extrapolation. Such an approach makes it possible not only to compare the mathematical properties of different approximations, but also to obtain an approximate estimate of age boundaries beyond which the brain's structure may deviate substantially from the familiar human norm. In this way, the study connects computational and conceptual levels of analysis, translating the original cultural-philosophical motif into a formalized framework.

Materials and Methods

The source material consisted of published estimates of synaptic density in the human frontal cortex [1] [1] for several age points: newborns, 1–2 years, 2–16 years, 16–72 years, and 74–90 years. For further analysis, representative values corresponding to the ages of 0.1, 2, 16, and 74 years were selected [1,4], as they make it possible to trace the overall trend of age-related changes and construct several approximation variants.

The numerical calculations were based on standard methods of regression analysis and interpolation. The compared models included linear, quadratic, cubic, exponential, Gaussian, and logarithmic functions [1,4]. To assess the quality of fit, the residual sum of squares (RSS), the coefficient of determination R^2, and the Akaike information criterion (AIC) were used, which makes it possible to compare both the accuracy of data description and the stability of model behavior during extrapolation.

Special attention was given to selecting a model suitable not only for interpolation within the observed range, but also for extrapolation beyond it. Since the aim of the study is not merely a formal description of the curve, but an estimate of threshold ages, the main selection criterion was the mathematical stability of the model outside the training interval. For this reason, linear regression was considered the basic model for the subsequent analysis.

The results were interpreted according to the following scheme: first, the approximation curves within the original range were compared; then their behavior was analyzed when extended into older ages; finally, threshold ages were calculated at which the function reached predetermined levels of synaptic density decline. This approach makes it possible to combine empirical neurophysiological data with a formalized estimate of the conditional age limit underlying the concept discussed in the text.

Table 1: Synaptic Density in the Frontal Cortex

Age group	Synaptic Density	Relative to Adult
Newborns	~11	100%
1–2 years (peak)	~16.6	150%
2–16 years (pruning)	16.6 → 11.05	150% → 100%
16–72 years (stability)	11.05 ± 0.41	100%
74–90 years (aging)	9.56 ± 0.28	~86%

Table 2: Heterochrony of Brain Maturation

Brain region	Synaptogenesis peak	Pruning completion
Auditory cortex	~3 months	~12 years
Middle frontal gyrus	>15 months	Mid-adolescence

Table 3: Synaptic Density in Pathology

Pathology	Change	Regions and correlation
Schizophrenia	Decrease (10–20%)	Prefrontal cortex, ACC; cognitive deficit
Depression / PTSD	Decrease	Dorsolateral prefrontal cortex, hippocampus
Autism	Increase	Sensory and associative cortical areas; social deficits
Alzheimer's disease	Decrease (up to 50%)	Hippocampus, parietal lobe; dementia

Table 4: Neurons and Synapses Across Species

Animal	Neurons (million)	Synapses (estimate)
Drosophila	140–150	~50–55 million
Mouse	71	10^12
Rat	200	~4.5 × 10^11
Cat	760	~10^13
Human	86,000	10^14

Table 5: Model Comparison

Model	RSS	R^2	AIC
Cubic polynomial	0.00	1.00	-183.86
Quadratic regression	17.73	0.39	23.31
Linear regression	18.86	0.35	21.55
Exponential model	17.98	—	—
Gaussian model	~0	—	—
Logarithmic model	26.44	—	—

Table 6: Interpolation of Function Values

Model	x=10	x=50	x=74	x=80
Cubic	22.14	—	9.56	98.71
Linear	—	—	9.35	9.03
Quadratic	—	10.92	9.54	11.74
Exponential	—	9.93	9.47	9.40
Gaussian	—	—	9.56	—
Logarithmic	—	11.23	11.10	11.08

Results and Discussion

The comparative analysis of approximation models showed a clear distinction between models optimized for interpolation and those more suitable for extrapolation. The cubic polynomial

provided an exact fit to the selected data points, as expected for an interpolating function, but its behavior outside the observed interval was unstable and therefore unsuitable for long-range prediction. By contrast, the linear model demonstrated a less precise fit within the sample but offered substantially greater stability when extended beyond the observed age range.

Among the models tested, the linear regression $P(x) = -0.05297x + 13.27$ yielded the most practical extrapolative behavior. According to this approximation, the synaptic density reaches the level y=2 at approximately 213 years, y=1.6 at about 220 years, and y=1.1 at roughly 230 years. These values may be interpreted as successive threshold zones marking a transition from the familiar human neural organization to a qualitatively altered state.

The quadratic model achieved only moderate explanatory power and produced a minimum near the middle of the age range, which does not correspond well to the empirical pattern of age-related synaptic decline. The exponential and Gaussian functions described specific local features of the data reasonably well but their asymptotic behavior did not support a biologically plausible long-range interpretation. The logarithmic model showed the weakest fit among the compared alternatives and was therefore of limited value for the present task [1,4].

From a methodological perspective, the comparison confirms that the choice of model must depend on the intended analytical purpose. If the aim is to reconstruct the internal structure of the observed data, then higher-order or symmetric functions may be preferable; if the aim is to obtain a stable extrapolation beyond the empirical interval, the linear model is the most defensible choice in this dataset. This distinction is crucial, because the article is not concerned with exact biological prediction but with the construction of a controlled quantitative analogy.

The derived thresholds should therefore be understood as heuristic rather than literal biological limits. They do not imply that synaptic density alone determines the complete fate of human consciousness or lifespan. Rather, they represent a formalized boundary within a simplified model, useful for illustrating how a culturally loaded idea such as the "200-year barrier" can be translated into a quantitative framework.

At the same time, the analysis highlights the limits of the present approach. The dataset is small, the age points are sparse [1,4], and the biological process of brain aging is influenced by many variables not included in the model, such as regional specificity, individual variability, pathology, and compensatory neuroplasticity. Consequently, the estimated age thresholds should be viewed as indicative model outputs rather than as predictions with direct clinical meaning.

Overall, the results support two conclusions. First, synaptic density declines with age in a way that can be approximated by multiple mathematical forms, each emphasizing different aspects of the trajectory. Second, when extrapolation is the primary goal, linear regression provides the most stable and interpretable basis for constructing a formal estimate of the age region associated with a marked decline in synaptic organization.

Conclusion

The present analysis suggests that synaptic density in the human frontal cortex [1] can be represented by several mathematical approximations, but the choice of model depends on the analytical objective. For interpolation within the observed age range, higher-order functions may provide a closer fit, whereas for extrapolation beyond the available data the linear model remains the most stable and transparent option.

Using the selected linear approximation, the model yields threshold ages of approximately 213, 220, and 230 years for successive levels of synaptic density decline. These values should not be interpreted as a biological forecast in the strict sense; rather, they define a formalized boundary within a simplified quantitative framework.

The principal contribution of the study is to connect neurophysiological data with a mathematically controlled discussion of the "200-year barrier." In this respect, the article demonstrates how a culturally loaded idea can be reformulated as a structured analytical model without eliminating its conceptual significance.

At the same time, the results must be interpreted with caution because the dataset is limited and the biological process of brain aging is multidimensional. Future work may improve the model by incorporating larger datasets, region-specific measures of synaptic density, and additional predictors of cognitive aging [4–14].

Acknowledgments

None.

Conflict of Interest

None.

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