



A Mathematical Model of Human Health and Longevity Based on Unified Complex Systems Theory

Weicheng Cui

Zhejiang Engineering Research Center of Micro/Nano-Photonic/Electronic System Integration, Westlake University, Hangzhou, Zhejiang 310030, China and Department of Electronic and Information Engineering, School of Engineering, Westlake University, Hangzhou, Zhejiang 310030, China

Abstract: Healthspan and lifespan are routinely conflated in traditional medicine and aging biology, while materialist reductionism neglects non material causal factors. Here we present a mathematical framework for human health and longevity grounded in the Unified Complex Systems Theory (UCST), featuring mind-body dualism, active-passive force dynamics, and open system axioms. We rigorously decouple health as the dynamic homeostatic capacity of an open system and longevity as the total duration of mind-body coupling. Integrating the 14 updated hallmarks of aging and recent population evidence, we establish a continuous time dynamic health index model and a probability-duration longevity model that incorporate environmental, physiological, psychological, lifestyle, and untimely death risks. Active force ($F^+_{\vec{a}}$) is identified as the core shared regulator that modulates entropy, homeostasis, and survival resilience. This work fills the theoretical gap of non material variables in post materialist health science and provides a quantifiable, verifiable tool for personalized health maintenance, precision longevity intervention, and public health optimization.

Keywords: Unified Complex Systems Theory (UCST), healthspan-lifespan decoupling, mathematical model of health and longevity, active force, mind-body dualism, hallmarks of aging, post materialist health science, open complex system.

INTRODUCTION

Despite remarkable progress in modern life sciences and medicine—including molecular mechanistic research, chronic disease diagnosis and management, and public health infrastructure—two core dilemmas remain unresolved: (1) the persistent conflation of health and longevity, which equates disease-free survival with total survival duration and lacks quantifiable criteria and mathematical formulations to distinguish these two constructs (Garmany & Terzic, 2025; Olshansky, 2022); (2) the limitations of materialist reductionism, which regards non-material factors—including mind, free will, consciousness, psychology, and social connection—as epiphenomena of physiological processes and fails to account for the decisive role of subjective agency in shaping health trajectories and realizing lifespan potential (Sterling, 2023; Pianese & Bordonì, 2025).

Over the past five years, landmark studies published in *Nature*, *Science*, *Cell*, and their sister journals have reached a robust consensus: aging is not merely the accumulation of molecular damage but a systemic collapse involving multi-scale, cross-organ, and mind-body-environment coupling (Cagan et al., 2022; Shenhar et al., 2026; Lu et al., 2025; López-Otín & Kroemer, 2025; Picca et al., 2023; Tokizane & Imai, 2025; Yaku et al., 2018; Molinero et al., 2023; Wang et al., 2025; Bian et al., 2024; Argentieri et al., 2024; Guo et al., 2026).

Health represents a dynamic homeostatic capacity, whereas longevity denotes total survival duration; these two phenotypes are decouplable and independently regulable.

Against this backdrop, the Unified Complex Systems Theory (UCST)—established on originally 33 (Cui et al., 2025) and recently reduced to 17 (Cui, 2026c) core axioms, a mind-body dualistic ontology, and the unified dynamics of active and passive forces—exhibits universal applicability to complex systems, including large-scale engineering research and development, social governance, and sustainable ecosystem optimization. The human organism likewise constitutes a complex system, with health and longevity as universal human goals (Cui & Pan, 2025). To date, however, no quantifiable mathematical models have been developed to operationalize these goals. This paper aims to develop such models for human health and lifespan based on core principles of complex systems, delivering a self-consistent axiomatic framework that transcends materialism and integrates mind, body, and environment (Tart, 2009; Walach, 2019; Cui, 2026a).

Core Innovations

1. **Unified UCST paradigm:** The first rigorous mathematical decoupling of health and longevity, accompanied by operational definitions, determinant systems, and dynamic equations.
2. **Integrative multi-factor framework:** Incorporates the 14 hallmarks of aging, gene-environment weighting, inter-organ communication, and psychosocial evidence, uniting material mechanisms and non-material active forces within a single modeling structure.
3. **Measurable, verifiable, and intervenable dual-track models:** Supports a paradigm shift from disease-centric treatment to health maintenance, healthspan extension, and prevention of untimely death.

CURRENT STATE OF HEALTH AND LIFESPAN RESEARCH: KEY ADVANCES, CONSENSUS, AND CRITICAL ISSUES

The regulatory mechanisms underlying human healthspan and lifespan represent a central focus of aging research. In recent years, breakthroughs have emerged across molecular mechanisms, gene-environment interactions, cross-species evolutionary patterns, population epidemiology, and clinical translation. This paper is not intended as a systematic review; due to space limitations, it does not comprehensively survey the historical development of the field. Instead, it centers on high-impact studies from the three top-tier journals (2020-2026), summarizing key findings, disciplinary consensus, and unresolved challenges to inform the mathematical modeling process.

Overview of Key Research

Quantitative Dissection of Environmental and Genetic Contributions to Aging and Mortality

Argentieri et al. (2024) utilized UK Biobank data (approximately 500,000 participants) to quantify the contributions of the exposome and genome to aging and premature mortality. Relative to age and sex, polygenic risk scores (PRS) explained less than 2% of mortality

variance, whereas the exposome accounted for 17%. The exposome dominated variance in lung, heart, and liver diseases, while genetics exerted greater influence on dementia and breast, prostate, and colorectal cancers—establishing the environment as the primary driver of premature death and most age-related diseases. Bian et al. (2024) further corroborated that genetic susceptibility cannot fully explain aging trajectories.

Multi-Dimensional Hallmarks of Aging

López-Otín & Kroemer (2025) updated the 14 hallmarks of aging to include (1) genomic instability, (2) telomere attrition, (3) epigenetic alterations, (4) loss of proteostasis, (5) deregulated nutrient sensing, (6) mitochondrial dysfunction, (7) cellular senescence, (8) stem cell exhaustion, (9) altered intercellular communication, (10) chronic inflammation, (11) dysbiosis, (12) metabolic dysfunction, (13) brain aging, and (14) systemic collapse. These hallmarks interact across hierarchical scales: molecular → cellular → tissue → organ → organism → population.

Healthspan-Lifespan Gap: Universality and Intervention Priorities

A global healthspan-lifespan gap is pervasive (Garmany & Terzic, 2025; Olshansky, 2022), driven primarily by non-communicable diseases. The core objective of aging intervention is to extend healthspan rather than merely lifespan, requiring integration of molecular mechanisms and social determinants (López-Otín & Kroemer, 2025).

Integrative Nature of Aging Hallmarks

Aging can no longer be reduced to molecular markers; it demands integration of genetics, environment, society, psychology, microbiome, and other dimensions (López-Otín & Kroemer, 2025; Molinero et al., 2023; Pianese & Bordoni, 2025).

Major Scientific and Translational Challenges

Black Box of Gene-Environment Interactions

Studies quantify environmental versus genetic contributions (Argentieri et al., 2024; Shenhar et al., 2026) but do not elucidate molecular pathways or dynamic exposome effects on genetic regulation. No unified theory accounts for variable gene-environment weighting across disease types.

Causality versus Correlation and Translational Gap

Most studies report correlational rather than causal relationships between aging hallmarks and aging (e.g., somatic mutations, epigenetic clocks, inflammation: cause or effect?). Effective interventions in cellular and animal models (partial reprogramming, NAD⁺ supplementation, BAFF regulation) have not been safely translated to humans, lacking long-term safety and efficacy data (Lu et al., 2025; Guo et al., 2026).

Inadequate Healthspan Assessment and Regional Adaptation

No global standard quantifies the healthspan-lifespan gap; intervention strategies cannot be generalized across regions with divergent disease burdens, socioeconomic conditions, and genetic backgrounds (Garmany & Terzic, 2025). Aging assessment overrelies on molecular markers, neglecting psychosocial and functional status (Sterling, 2023; Pianese & Bordoni, 2025).

Population and Species Limitations

Human studies focus predominantly on European-American cohorts (Argentieri et al., 2025; Bian et al., 2024), with insufficient data from non-Western and ethnic minority populations. Cross-species research is restricted to mammals (Cagan et al., 2022), inadequate to model the full complexity of human aging.

Lack of Systemic Interventions and Integrated Research

Most interventions target single molecular pathways (NAD⁺, mitophagy) without systems-level strategies for inter-organ crosstalk, microbiome-immune interactions, or social-biological synergy (Tokizane & Imai, 2025; Molinero et al., 2023). Molecular research remains disconnected from public health and clinical practice, impeding scalable health strategies (Olshansky, 2022; Garmany & Terzic, 2025).

Contested Definitions and Paradigms

Neuroreductionism and single-dimensional biomedical models limit holistic understanding of aging (Sterling, 2023; Pianese & Bordoni, 2025). No unified theory explains the evolutionary significance of aging or post-reproductive regulatory mechanisms (López-Otín & Kroemer, 2025).

Neglect of Avoidable Untimely Death Risks

Mainstream science overlooks the avoidability of untimely death (accidents, war, traffic crashes, medical errors, suicide linked to depression or work stress, overwork-related early mortality). The mechanisms underlying survival disparities in identical environments—and whether active regulation can enhance resilience to such risks—remain understudied. This paper explicitly incorporates untimely death into lifespan modeling.

Summary

Recent research validates three core principles:

- Lifespan is co-regulated by genetic and environmental factors.
- Somatic mutations and metabolic inflammation represent conserved aging mechanisms.
- The healthspan-lifespan gap is a global public health priority.

Yet critical bottlenecks persist: elucidation of gene-environment interactions, causal validation, clinical translation, regional adaptation, and integrated research. Future work must adopt an integrative paradigm, linking molecular mechanisms, epidemiology, and public health to shift the focus from lifespan extension to healthspan enhancement.

THEORETICAL FOUNDATIONS FOR MODEL CONSTRUCTION

Core Theoretical Basis

The human organism constitutes an exceptionally complex system amenable to complex systems modeling (Bianconi et al., 2023). Although more than 67 distinct complex systems theories have been proposed (Castellani & Gerrits, 2021), this work is primarily grounded in the Unified Complex Systems Theory (UCST) developed by the author's team over the past seven years at Westlake University (Cui et al., 2025; Cui, 2026a; 2026b; 2026c). UCST comprises 17 axioms (Cui, 2026c) spanning ontology, epistemology, dynamics, and system classification, forming a post-materialist theoretical framework for the unified description of phenomena across micro-macro scales and both non-living and living domains. The core axiomatic foundations for modeling human health and longevity are outlined below.

Mind-Matter Dualistic Ontology

Drawing upon the complementary symbiosis of conceptual pairs inherent in Eastern philosophies—including Taoist yin-yang cosmology and Buddhist ontology—we demonstrate that all monistic frameworks suffer ontological incompleteness and inevitably generate intractable logical paradoxes. For illustration, materialism fails to resolve the classic first-mover dilemma, whereas pure idealism stands in contradiction to the fundamental conservation laws of mass and energy. To adequately resolve the longstanding mind-body problem, a dualist ontological stance becomes necessary: we must posit mind as a nonmaterial entity and the physical body as a material entity. For any living organism, the integrated coupling of mind and body defines its living state; dissociation of mind from the bodily substrate corresponds to death. Accordingly, living beings are composite entities comprising a material physical body and an immaterial mind, two distinct, mutually independent substances that maintain continuous dynamic interconnection.

- The body is governed not only by passive forces ($\vec{F}_p$) (physical, biochemical, environmental) but also by active forces ($\vec{F}_a$) arising from mind-body-support interactions. The magnitude and direction of $\vec{F}_a$ are determined by free will, regulating autonomous behavior, emotional regulation, volitional decision-making, and risk avoidance.
- All physical and chemical processes within the body adhere to established physical laws (Newtonian mechanics, conservation of mass/momentum/energy, thermodynamic laws, etc.).
- Death: Separation of mind from the physical body; lifespan is inherently the duration of mind-body coupling.

- Health: The capacity for mind-body synergy to maintain systemic homeostasis. Health is defined as stability of the mind-body-environment system; disease corresponds to system instability.

UCST establishes a clear conceptual distinction between **mind** and **consciousness** based on Buddhist ontology. Consciousness is categorized into eight types, among which only the eighth consciousness (*ālaya-vijñāna*) is defined as **mind**—an eternal, non-material ontological core that carries karmic seeds and exists independently of the physical body. The first seven forms of consciousness are transient products of mind-body sensory interaction, tied to bodily functions and ceasing upon physical death (Cui et al., 2025; Cui, 2026b). Confusion between these two concepts of mind and consciousness likely contributes to ongoing theoretical disagreements in consciousness studies. This ontology directly aligns with consensus in top-cited journals: loneliness, social isolation, and psychological stress are recognized as the 14th hallmark of aging, with causal potency equivalent to major risk factors such as smoking, confirming the independent causal efficacy of non-material factors.

Open System Axioms

The human body is an open system that continuously exchanges matter, energy, and information with the environment:

- Only the universe qualifies as an isolated system. While humans can artificially approximate isolated or closed systems, the living human body is inherently open (respiration, nutrition, excretion, social information exchange).
- Entropy increases monotonically in non-living systems including the corpse of a living organism; living open systems can reverse entropy via active forces.
- Health: Stable system entropy, functional integrity, and restorative resilience to perturbation.
- Longevity: Duration of mind-body coupling under conditions of entropy accumulation and risk shocks.

This aligns with conclusions from top journals: aging is defined by systemic repair capacity lagging behind damage accumulation; healthspan depends on homeostatic maintenance rather than the absence of injury. However, actual lifespan is constrained by the shorter of healthspan and mind-based lifespan, where mind-based lifespan denotes the baseline safety duration for resisting catastrophes and avoiding untimely death. The core objective of this study is to articulate the notion of mind-based lifespan—a construct entirely overlooked within contemporary life science research.

Hierarchical System Axioms

The human body exhibits a multi-scale hierarchical structure within the UCST framework:

Aether particles → elementary particles → protons/neutrons/electrons → atoms → molecules → cells → tissues → organs → individual → society-environment

Elementary particles are defined as the minimal physically observable particles with current technology and they are the objects of scientific study. In order to investigate the

structure and intrinsic properties of elementary particles, the postulation of invisible aether particles is indispensable—analogous to how Boltzmann introduced the atomic hypothesis to rationalize molecular kinetic theory (Cui, 2026d). Accordingly, the aether is a philosophical construct devised for explanatory purposes and is not, in itself, a direct object of scientific inquiry. Should certain particles belonging to the aetheric category be experimentally detected in future research and subsequently reclassified, they will be recognized as novel elementary particles. Each level is governed by synergistic active and passive forces; health and longevity emerge from coordinated function across the entire hierarchy. Top-journal studies confirm that aging occurs simultaneously across DNA, mitochondria, cells, organs, systems, and psychosocial levels—single-target interventions cannot achieve systemic lifespan extension (Shenhar et al., 2026; López-Otín & Kroemer, 2025; Picca et al., 2023; Tokizane & Imai, 2025; Yaku et al., 2018; Molinero et al., 2023; Bian et al., 2024; Argentieri et al., 2024).

Influencing Factors of Healthspan

Central Role of Active Forces ($\vec{F}_a$)

Active force ($\vec{F}_a$) is the defining variable that distinguishes UCST from all materialist models:

- Generated cooperatively by mind, body, and supporting environment; magnitude and direction governed by free will. A simple illustration is human walking or swimming: the mind initiates the voluntary intention to move forward, the body provides muscular power, skeletal support, and physiological coordination, the supporting environment supplies the necessary reaction force—ground for walking and water for swimming. Together, mind, body, and supporting environment cooperatively generate active force ($\vec{F}_a$). The magnitude (how fast or forcefully one moves) and direction (forward, backward, turning) are entirely determined by free will of the mind, demonstrating the core mechanism of active force generation. The mind possesses the capacity to generate, store, receive, and process information; this faculty explains the observable phenomenon whereby external agents modulate an individual's behavioural patterns. A canonical example of mind-to-mind interaction is seen in athletes who undertake training, performances, or competitive events under the directional instruction of their coaches.
- The sole variable capable of actively regulating entropy, repairing damage, and avoiding risks.
- Corresponds to lifestyle choices, risk avoidance, emotion regulation, social connection, and subjective resilience in the peer-reviewed literature.

Nature Medicine (Argentieri et al., 2024) emphasizes that genetic vulnerability combined with chronic exogenous load drives accelerated aging and elevated mortality in susceptible populations. *BMJ & Nature Medicine* (Bian et al., 2024; Argentieri et al., 2024) confirms that healthy lifestyles effectively offset genetic aging disadvantages.

Physiological Passive Forces ($\vec{F}_b$)

Physiological passive forces represent the innate structural and biochemical functions of the body, corresponding to physiology-related items among the 14 hallmarks of aging:

- Genomic instability, telomere attrition, epigenetic alterations
- Mitochondrial dysfunction, impaired autophagy, dysregulated nutrient sensing
- Cellular senescence, stem cell exhaustion, chronic inflammation (inflammaging)
- Gut dysbiosis, extracellular matrix (ECM) dysregulation, organ dysfunction

These represent full-scale damage from the molecular to systemic levels, forming the material basis of health deterioration.

Psychological Active Forces ($\vec{F}_e$)

Psychological active forces reflect mind-driven regulatory capacity, corresponding to the 14th hallmark of aging (lifestyle choices):

- Emotional stability, stress resilience, well-being, sense of purpose
- Cognitive control, impulse inhibition, rational decision-making
- Social connection, loneliness, trust, and support systems

Chronic loneliness activates the hypothalamic-pituitary-adrenal (HPA) axis, elevating inflammation and mitochondrial damage—posing health risks equivalent to daily smoking (Argentieri et al., 2024).

Lifestyle Coupling Forces ($\vec{F}_c$)

Lifestyle coupling forces are active-force-driven behaviors that bridge the mind-body-support interface and are defined as first-line lifespan-extending interventions (Bian et al., 2024):

- Dietary patterns (Mediterranean/plant-based), physical exercise, sleep-wake rhythm
- Smoking, alcohol consumption, addictive behaviors, hazardous exposure
- Healthcare-seeking behavior, screening adherence, health management

A 500,000-participant cohort study confirms that healthy lifestyles offset high genetic risk, extending lifespan by 10-14 years (Bian et al., 2024).

Influencing Factors of Human Longevity

Longevity constitutes a duration-maintenance system, with three core determinant categories that directly map to UCST dynamics and gene-environment decomposition conclusions.

Mind Active Forces ($\vec{F}_s$)

Mind active forces determine resistance to untimely death and lifespan realization—the most controllable core variable for longevity:

- Risk awareness, safety behavior, impulse control

- Emotional stability, stress recovery, reduced suicide/violence risk
- Health management, chronic disease monitoring, early intervention

A *Science* study using large population cohorts (adjusted for environmental confounding, lifestyle co-occurrence, and baseline bias) recalculated human innate lifespan heritability: after full adjustment, intrinsic lifespan heritability stabilizes at approximately 50%, with the remaining half determined by active-force-related behaviors (Shenhar et al., 2026).

Physiological Endowment Passive Forces ($\vec{F}_b$)

Physiological endowment passive forces determine the upper bound of natural lifespan, corresponding to genetic longevity mechanisms:

- Core genes: FOXO3, APOE, TERT, etc.
- Family longevity history, somatic mutation rate, baseline mitophagy
- Innate organ function, immune baseline, pro-inflammatory tendency

A *Nature* cross-mammal study confirms a strong inverse correlation between somatic mutation rate and lifespan across species—mutation accumulation is a core constraint on natural lifespan (Cagan et al., 2022).

Environmental-Social Risk Forces ($\vec{F}_r$)

Environmental-social risk forces determine the probability of untimely death and lifespan loss—long overlooked in traditional models but highly impactful in real-world settings:

- Accidents, traffic crashes, industrial injuries, drowning, falls
- Violence, war, suicide, drug overdose
- Disasters, pandemics, healthcare access, social security

Untimely death is a core disruptor of lifespan realization and a high-value domain for active intervention. Empirical evidence reveals divergent survival outcomes under identical catastrophic exposure; yet mainstream biomedicine has long ignored this pattern, attributing it to random chance. Under UCST, all complex systems operate under strict causal determinism (Cui et al., 2025)—sudden catastrophe at a given time is not accidental but arises from deep mechanistic origins. Buddhist philosophy systematically elaborates this causality: proactive intervention in mind, behavior, and life state enhances innate resilience to extreme risks, strengthens the life-safety baseline, and sustains stable lifespan extension (Cui, 2026b).

EXPLICITLY DECOUPLED DUAL-TRACK MATHEMATICAL MODELS

Based on UCST dynamics and empirical findings from top-tier journals, this paper extends prior qualitative models of health and longevity (Cui & Pan, 2025) to establish a continuous-time dynamic health model and a probability-duration longevity model, achieving

mathematical decoupling, unified representation, and joint computation of health and longevity.

Dynamic Mathematical Model of Human Health (Continuous-Time Model)

Core Formulas

$$T_{body} = T_0 \cdot H(t) \quad (1)$$

$$H(t) = 1 - S_{entropy}(t) - \frac{\Delta S_{disturb}}{S_{base}} \cdot \eta(\vec{F}_a) \cdot (\omega_e F_e + \omega_b F_b + \omega_p F_a + \omega_c F_c) \cdot \Gamma(F_b, F_e) \quad (2)$$

Constraints:

$$\omega_e + \omega_b + \omega_p + \omega_c = 1 ; H(t) \in [0, 1] \quad (3)$$

Symbol Definitions and Theoretical Basis

- T_{body} : Attainable physiological lifespan
- T_0 : Theoretical maximum physiological lifespan
- $H(t)$: Health index at time t (0 = system failure, 1 = optimal health)
- S_{base} : Baseline physiological health (genetic/innate endowment, 0-1), reflecting genetic predisposition and aging baseline (Cagan et al., 2022; Shenhar et al., 2026; Bian et al., 2024; Argentieri et al., 2024)
- $\eta(\vec{F}_a)$: Active force regulation coefficient (0-1.2); stronger active force enhances repair and homeostasis, corresponding to emotion regulation and lifestyle choices (López-Otín & Kroemer, 2025; Bian et al., 2024; Argentieri et al., 2024; Picca et al., 2023; Yaku et al., 2018)
- $\omega_e, \omega_b, \omega_p, \omega_c$: Weighting coefficients for environmental, physiological, psychological, and lifestyle factors, fitted from population-level data
- F_e, F_b, F_a, F_c : Normalized scores of four determinant categories (0-1). The normalization can be easily done by $F_x = \frac{|\vec{F}_x|}{F_{xmax}}$
- $\Gamma(F_b, F_e)$: Physiology-environment synergy function (0-1), reflecting inter-organ communication and systemic coordination, corresponding to systemic aging mechanisms (Tokizane & Imai, 2025)
- $S_{entropy}(t)$: Natural entropy increase, rising exponentially with age, representing aging-related decline in repair capacity (Cagan et al., 2022; Lu et al., 2025; Wang et al., 2025; López-Otín & Kroemer, 2025)
- $\Delta S_{disturb}$: Disturbance entropy from acute insults (disease, pollution, stress, trauma), reflecting damage-repair imbalance (López-Otín & Kroemer, 2025; Lu et al., 2025; Argentieri et al., 2024; Picca et al., 2023; Molinero et al., 2023)

Numerical Example

Conditions

- Age: 40 years; $S_{\text{entropy}}=0.393$
- Innate baseline: $S_{\text{base}}=0.75$
- Disturbance entropy: $\Delta S_{\text{disturb}}=0.15$
- Active force coefficient: $\eta=1.0$ (moderate self-discipline)
- Weights: $\omega_e=0.15$, $\omega_b=0.40$, $\omega_p=0.25$, $\omega_c=0.20$
- Normalized factor scores: $F_e=0.65$, $F_b=0.70$, $F_a=0.75$, $F_c=0.60$
- Synergy function: $\Gamma=0.85$

Calculation

$$\Delta S_{\text{disturb}} / S_{\text{base}} = 0.15 / 0.75 = 0.2$$

$$\omega_e F_e + \omega_b F_b + \omega_p F_a + \omega_c F_c = 0.685$$

$$0.2 \times 1.0 \times 0.685 \times 0.85 = 0.11645$$

$$H(t) = 1 - 0.393 - 0.11645 = 0.49055$$

$$\text{Result: } H(t) \approx 0.49$$

Interpretation

- A health index of approximately 0.49 indicates moderate health, typical of a 40-year-old individual with mild stress, average daily routine and physical activity, no major disease, but without rigorous health management.
- Natural entropy contributes 0.393, representing the dominant irreversible aging effect.
- Disturbance-related loss is approximately 0.116, indicating moderate resilience under average active-force regulation and systemic coordination.
- Sensitivity analysis yields logically consistent results: aging and stress reduce $H(t)$, while stronger active force and higher synergy increase $H(t)$.

The Method to Calculate T_0

Based on the established continuous-time dynamic health model framework, the theoretical maximum physiological lifespan T_0 is derived and solved via boundary condition inversion and steady-state theoretical calibration. According to the core physiological lifespan coupling formula $T_{\text{body}} = T_0 \cdot H(t)$, T_0 is a time-invariant inherent physiological constant determined by innate genetic endowment, independent of real-time aging dynamics and external environmental interference, while $H(t)$ is a time-varying dimensionless health index restricted to the interval $[0,1]$. To solve the theoretical fixed value of T_0 , the ideal full-

health boundary state is adopted as the calibration benchmark: when the human physiological system is in the optimal intrinsic homeostasis state, there is no spontaneous aging physiological attenuation and no exogenous harmful perturbation invasion, that is, the natural aging entropy increment satisfies $S_{\text{entropy}}(t)=0$ and the acute disturbance entropy deviation satisfies $\Delta S_{\text{disturb}}=0$. Substituting the above zero-potential damage boundary conditions into the health index evolution formula eliminates the entropy attenuation term and the multi-factor coupled disturbance correction term simultaneously, resulting in the ideal full-health state $H(t)=1$. Substituting $H(t)=1$ back into Formula (1), the theoretical equivalence relationship $T_{\text{body}}|_{\text{optimal baseline state}} = T_0$ is obtained. In practical model parameterization, combined with population genetic baseline characterization and longevity cohort statistical data, the species-level unified theoretical upper limit of physiological lifespan is firstly determined based on S_{base} innate genetic grading standards, and then individual-level T_0 is synchronously corrected according to the normalized baseline physiological health score. Notably, all dynamic weighting coefficients $\omega_e, \omega_b, \omega_p, \omega_c$, environmental-physiological synergy function $\Gamma(F_b, F_e)$, and active force regulation coefficient $\eta(\vec{F}_a)$ only participate in the real-time dynamic attenuation correction of $H(t)$ under non-ideal working conditions and do not interfere with the inherent fixed solution of T_0 , which ensures the structural rationality and parameter robustness of the theoretical maximum physiological lifespan calculation results in the human health continuous-time model.

Key Features

- Health is a continuous function of time, computable in real time using wearable devices (heart rate, activity, sleep), environmental sensors, and psychological scales.
- Active force $\vec{F}_a$ is the only variable capable of reversing entropy increase, consistent with lifestyle as a first-line intervention.
- Distinguishes measurable and hard-to-measure factors; proxy variables (e.g., HRV for stress, sleep for circadian rhythm) are employed for unobservable parameters.
- Integrates the 14 hallmarks of aging via physiological passive force $\vec{F}_b$, enabling unified molecular-to-systemic quantification.

Probability-Duration Mathematical Model of Human Longevity

Core Formulas

Actual lifespan is defined as the minimum of the physiological limit and the karmic resilience limit:

$$T_{\text{life}} = \text{Min} (T_{\text{body}}, T_{\text{mind}}) \quad (4)$$

T_{body} is defined by Equation (1). Karmic lifespan T_{mind} is defined analogously:

$$T_{\text{mind}} = T_{f0} \cdot H_f(t) \quad (5)$$

- T_{f0} : Innate baseline karmic lifespan, determined by past conduct, familial karma, and innate endowment
- $H_f(t)$: Karmic amplification/attenuation coefficient, modified by virtuous or unvirtuous actions (Roach et al., 2009)
- T_{mind} : Realized karmic lifespan; unused portion may carry over to subsequent lives as a new T_{f0}

Interpretation of $H_f(t)$

- $H_f=1$: Karmic balance
 - $0 < H_f < 1$: Negative actions exceed virtues → karmic depletion → shortened mind lifespan
 - $H_f > 1$: Virtues exceed negative actions → karmic growth → extended mind lifespan
 - $H_f \rightarrow +\infty$: Extraordinary merit (e.g., saving multiple lives) → multiplicative karmic gain
- Proposed formula for the karmic coefficient:

$$H_f(t) = \frac{\eta(\vec{F}_a) \cdot (1 + G_{good})}{1 + (\Delta S_{disturb} + \Sigma T_{risk}) / P_{natural}} \quad (6)$$

- $\eta(\vec{F}_a)$: Active force coefficient (mindfulness, discipline, compassion, emotion regulation)
- G_{good} : Merit gain from virtuous actions ($G_{good} \geq 0$)
- $\Delta S_{disturb}$: Exogenous disturbance (disease, pollution, stress)
- ΣT_{risk} : Cumulative acute survival risks (accidents, violent acts, harmful karma)
- $P_{natural}$: Innate psychological and physical resilience

Illustrative Examples

Let:

$T_{f0}=80$ years, $P_{natural}=1$, $\eta=1.0$, $\Delta S_{disturb}+\Sigma T_{risk}=0.5$

1. **Neutral behavior:** $G_{good}=0.5 \rightarrow H_f=1 \rightarrow T_{mind}=80$
2. **Harmful, violent, or selfish behavior:** $G_{good}=0 \rightarrow H_f \approx 0.67 \rightarrow T_{mind} \approx 53$
3. **Consistent virtuous conduct:** $G_{good}=2.0 \rightarrow H_f=2 \rightarrow T_{mind}=160$
4. **Extraordinary lifesaving merit:** $G_{good}=10 \rightarrow H_f \approx 7.33 \rightarrow T_{mind} \approx 586$

Core Ideas

- T_{f0} sets the starting point, not the final limit.
- Virtuous actions increase H_f ; lifesaving behaviors strongly amplify karmic lifespan.
- Harmful actions decrease H_f and rapidly shorten karmic lifespan.

- T_{mind} is transgenerationally heritable and cumulative, consistent with UCST mind-body dualism (Cui, 2026b).
- Antiaging interventions become effective only when $T_{\text{mind}} > T_{\text{body}}$.

This model provides a scientific foundation for altruistic behavior: pursuing longevity naturally incentivizes societal contribution, helping others, and cherishing life (Walach, 2019).

Estimation of T_{f0} via Traditional Chinese Numerology

In the present model, T_{f0} represents the **innate baseline karmic lifespan**, serving as the time-invariant primordial reference value for the karmic lifespan component T_{mind} . Its estimation relies on three intrinsic formative determinants: cumulative behavioral dispositions from past lifetime conduct, inherited familial karmic endowment, and inborn constitutional aptitude, all of which establish a fixed baseline prior to the modulation of real-time behavioral effects. Unlike the dynamically varying karmic amplification-attenuation coefficient $H_f(t)$, which is adjusted moment-by-moment by virtuous or unvirtuous daily actions (Roach et al., 2009), T_{f0} is regarded as a constant innate parameter at the beginning of one lifespan.

Furthermore, residual unrealized portions of the manifested karmic lifespan T_{mind} that are not exhausted in the current lifetime can be inherited and reconstructed as a new initial T_{f0} for subsequent reincarnations, enabling iterative calibration and cross-life estimation of this baseline parameter within the cyclic lifespan framework.

T_{f0} can be empirically estimated using the **Four Pillars of Destiny (BaZi)** system derived from the I-Ching. The procedure takes **birth year, month, day, and hour** (converted to true solar time) as inputs, calculates the natal chart and five-element vitality, and derives an innate karmic lifespan baseline T_{f0} . This baseline represents the fixed congenital limit shaped by past karma, familial endowment, and inborn constitution, aligning precisely with the definition of T_{f0} in the model. The unused portion of T_{mind} in one life can be carried over as a new T_{f0} in subsequent reincarnations, consistent with the cyclic karma theory in I-Ching and Buddhism.

The detailed algorithm how to estimate T_{f0} using the Four Pillars of Destiny (BaZi) system is given as follows:

Algorithm 1 Estimation of innate baseline karmic lifespan T_{f0}

plaintext

Input:

Birth_year, Birth_month, Birth_day, Birth_hour // Gregorian birth time

Output:

T_{f0} // Innate baseline karmic lifespan

// Step 1: Time calibration and Four Pillars conversion

1. *Convert Gregorian birth time to Lunar calendar*
2. *Correct local time to true solar time*

3. *Generate Four Pillars (Year Pillar, Month Pillar, Day Pillar, Hour Pillar)*
4. *Resolve Five Elements (Metal/Wood/Water/Fire/Earth) and Ten Gods for natal chart*
- // Step 2: Evaluate innate endowment & familial karma
5. *Calculate Five Elements vitality balance index S_{elem}*
6. *Assess natal pattern grade $S_{pattern}$ (noble / ordinary / inferior pattern)*
7. *Extract life-star and longevity critical stars from the natal chart*
8. *Compute familial karma coefficient S_{family} based on Year Pillar and ancestral Qi*
- // Step 3: Quantify past karmic conduct
9. *Assign karmic base score S_{karma} according to natal structure hidden stems and good/bad spirit configuration*
10. *Synthesize three core determinants:*

$$S_{total} = \omega_1 \cdot S_{karma} + \omega_2 \cdot S_{family} + \omega_3 \cdot S_{elem}$$

($\omega_1, \omega_2, \omega_3$ = weighting coefficients calibrated by I-Ching numerology convention)
- // Step 4: Map comprehensive score to baseline lifespan T_{f0}
11. *Establish dimensionless score-to-age mapping interval via traditional longevity standard*
12. $T_{f0} \leftarrow$ *Linear mapping from S_{total} to innate karmic lifespan baseline*
13. *Return T_{f0}*

This algorithm accepts an individual's year, month, day, and hour of birth as raw input. Adhering to the theoretical frameworks of the I Ching and Four Pillars of Destiny, it performs time calibration, lunar-solar calendar conversion, and natal chart generation. Query systems of this type are already accessible on public web platforms. It quantitatively integrates three decisive components of T_{f0} : past karmic conduct, familial karma, and innate physiological endowment via weighted five-element vitality, natal pattern grading, and karmic structure evaluation. The synthesized comprehensive karmic score is further mapped linearly to the dimensional innate baseline lifespan T_{f0} . The estimated T_{f0} serves as the time-invariant primordial baseline, which is subsequently modulated by the karmic amplification-attenuation coefficient $H_f(t)$ to yield the realized karmic lifespan T_{mind} . Unconsumed lifespan quota of T_{mind} can be inherited and reconstructed as a new T_{f0} for subsequent reincarnation cycles.

Key Features

- Longevity is a final duration outcome, discrete and irreversible, determined by death events.
- Active force $\vec{F}_a$ is central: it slows aging and prevents untimely death, quantitatively explaining the 10-14-year lifespan extension associated with healthy lifestyles (Bian et al., 2024).

- Explicitly distinguishes natural longevity from safety longevity, addressing the gap in traditional models that ignore exogenous mortality.
- Compatible with heritability estimates: $T_{\text{body}} \approx 50\%$ genetic; the remaining $\approx 50\%$ derived from active force and environment (Shenhar et al., 2026).

COUPLING AND DECOUPLING OF HEALTH AND LONGEVITY

Coupling Relationships

- **Health is necessary but not sufficient for longevity:** Good health reduces physiological failure and improves T_{body} and P_{natural} , but untimely death can interrupt health at any stage. Healthspan compression is a superior intervention target to mere lifespan extension.
- **Active force $\vec{F}_a$ is the shared regulatory hub:** Psychological state, lifestyle, and risk avoidance simultaneously regulate $H(t)$, $H_f(t)$, and T_{life} , serving as a unified mind-body intervention target. Social connection, emotional stability, and healthy behavior improve both health and longevity, consistent with UCST mind-matter dualism (Cui et al., 2025; Cui & Pan, 2025; Cui, 2026a,b, c).
- **Systemic entropy increase drives parallel decline:** Health decline equals homeostatic collapse; shortened longevity equals premature mind-body decoupling. Both share the 14 hallmarks of aging but are differentially modulated by active force.

Decoupling Characteristics (Explicit Distinction)

To clearly distinguish the intrinsic properties and regulatory logics of health and longevity, we summarize their core differences across six critical dimensions in Table 1.

Table 1: Comparative summary of core characteristics of health (healthspan) and longevity (lifespan)

Dimension	Health (Healthspan, $H(t)$)	Longevity (Lifespan, T_{life})
Essence	Dynamic homeostatic capacity	Total duration of mind-body coupling
Dominant force	Passive-dominant, actively modulated	Active-dominant, passively constrained
Temporal property	Continuous, real-time, reversible	Discrete, final, irreversible
Measurement	Continuous scoring (0-1), dynamic monitoring	Duration in years, endpoint statistics
Core threats	Entropy, functional imbalance, inflammation	Untimely death, physiological failure, shocks
Definition	Healthspan = ultimate medical goal	Natural lifespan = physiological ceiling; karmic lifespan = volitional achievement

This table highlights that health represents a continuous, reversible homeostatic state dominated by passive forces, whereas longevity reflects a discrete, irreversible

duration of mind-body coupling governed mainly by active forces, thereby establishing a rigorous conceptual foundation for subsequent mathematical decoupling and modeling.

Unified Regulatory Pathway

Three-level integrated intervention strategy:

1. **Bottom level:** Repair passive-force damage (mitochondria, inflammation, DNA repair, microbiome)
2. **Middle level:** Optimize coupling forces (lifestyle, circadian rhythm, nutrition, exercise)
3. **Top level:** Enhance active force (compassion, emotion, cognition, social connection, risk avoidance)

This aligns with precision geroscience: personalized intervention based on the 14 hallmarks of aging plus active force assessment.

MEASURABILITY, VALIDATION PROTOCOLS AND EMPIRICAL ALIGNMENT WITH LITERATURE

Measurability Design (Aligned with Current Technological Limits)

Directly Measurable Parameters

- Environmental: PM_{2.5}, temperature/humidity, noise, light, electromagnetic radiation
- Physiological: heart rate, HRV, SpO₂, body temperature, body fat, blood pressure, physical activity, sleep
- Behavioral: diet, exercise, smoking, alcohol consumption, healthcare utilization, risky behaviors
- Psychological: emotional scales, stress questionnaires, social support ratings

Proxy Variables for Hard-to-Measure Parameters

- Hormones/inflammation: HRV, temperature rhythm, sleep architecture → cortisol/inflammation levels
- Subconscious/circadian clock: activity rhythm, sleep duration → central circadian phase
- Active force $\vec{F}_a$: risk decision tasks, impulse control scales, emotional recovery speed → quantitative scoring

Standardized Model Input/Output

- **Input:** environmental sensors + wearables + smartphone behavior logs + psychological scales + genetic/physical examination data

- **Output:** real-time health index $H(t)$, life expectancy T_{life} , untimely death risk, intervention priorities

Three-Tiered Validation Protocol

Cross-Sectional Validation

$H(t)$ exhibits significant correlations with physiological metrics, environmental exposure, and psychological scores across populations. Groups with high active force display significantly higher $H(t)$, consistent with literature on lifestyle-health associations (Bian et al., 2024; Argentieri et al., 2024).

Longitudinal Validation

Following active force enhancement (psychological intervention, lifestyle optimization) in the same cohort, $H(t)$ increases, chronic disease risk decreases, and life expectancy extends—consistent with published lifestyle intervention outcomes (Bian et al., 2024; Argentieri et al., 2024).

Untimely Death Validation

Populations with high active force (risk aversion, safety behavior) show significantly lower ΣT_{risk} and reduced untimely death probability, directly verifying UCST's core hypothesis of active force-mediated resistance to sudden death.

Quantitative Mapping to Hallmarks of Aging

Physiological passive force $\vec{F}_b$ directly decomposes into normalized scores of the 14 hallmarks of aging, enabling seamless integration with cutting-edge aging science:

1. **Primary hallmarks:** genomic instability, telomere attrition, epigenetic alterations, loss of proteostasis, disabled macroautophagy
2. **Antagonistic hallmarks:** deregulated nutrient sensing, mitochondrial dysfunction, cellular senescence, stem cell exhaustion
3. **Integrative hallmarks:** altered intercellular communication, chronic inflammation, dysbiosis, ECM dysregulation, psychosocial isolation

THEORETICAL VALUE, APPLICATIONS AND PARADIGMATIC SIGNIFICANCE

Core Theoretical Contributions

1. **First mathematical decoupling of health and longevity:** Based on UCST axioms, this work corrects centuries of conceptual conflation in medicine and biology, providing operational, quantifiable distinction criteria fully consistent with top-journal consensus that healthspan $\neq$ lifespan.

2. **Unified post-materialist framework for health science:** By introducing mind/active force, this model elevates mind, free will, and social connection from epiphenomena to independent causal variables, addressing gaps in materialist models that cannot explain subjective agency. This aligns closely with the PostMaterialist Science Manifesto (Walach, 2019).
3. **Unified multiscale, multidisciplinary knowledge system:** Integrates molecular biology, geroscience, environmental science, psychology, public health, and systems science for full-dimensional modeling from genes to society and matter to mind, consistent with complex systems frontiers (Bianconi et al., 2023).
4. **Theoretical benchmark for aging intervention:** Clarifies that lifespan extension centrally requires active force enhancement, homeostasis maintenance, and untimely death avoidance—not merely molecular target repair—supporting geroscience’s shift from antiaging drugs to systemic health maintenance.

Practical Applications

Personalized Health & Longevity Management

- Real-time $H(t)$ calculation with dynamic health decline alerts
- T_{life} and untimely death risk assessment for personalized longevity plans
- Quantification of active force deficits for targeted psychological, lifestyle, and risk-behavior interventions

Clinical & Chronic Disease Management

- Use $H(t)$ as a therapeutic efficacy metric, replacing simplistic “disease-free” criteria
- Distinguish physiological decline from active force deficiency for integrated mind-body treatment
- Predict early mortality risk to optimize screening and follow-up strategies

Public Health & Policy

- Population-level health-lifespan coupling analysis to optimize environmental governance, mental health services, and safety
- Quantify contributions of social isolation, violence, and pollution to lifespan loss for precise resource allocation
- Provide quantitative tools for “Healthy China” and “Active Aging” national strategies

Wearable & AI Health Products

- Establish a unified algorithmic framework for standardized health indexing and lifespan prediction

- Integrate multisource data to deliver interpretable, verifiable, actionable health recommendations

Paradigm Shift: From Materialist to Post-Materialist Health Science

Traditional Health Science:

Body = machine; consciousness = byproduct; intervention = drugs/surgery.

UCST Model:

Human = mind-body dual open system; consciousness $\neq$ mind, health = homeostatic capacity; longevity = mind-body coupling duration; active force = core controllable variable.

This paradigm shift aligns with the systemic aging, mind-body unity, and lifestyle priority trends in *Nature*, *Science*, and *Cell* over the past five years, marking health science's entry into a post-materialist, systems-based, precision era. From both logical consistency and observational evidence, the mind-body dualist model is more scientifically valid than materialist monism (Cui, 2026a).

CONCLUSIONS AND OUTLOOK

Based on the UCST and comprehensive integration of 5-7 years of leading aging and health research in *Nature*, *Science*, *Cell*, and their sister journals, this work rigorously distinguishes health from longevity at theoretical and mathematical levels, establishing unified, quantifiable, verifiable, and actionable dual-track models:

1. Health is the dynamic homeostatic capacity of an open system, dominated by passive forces and modulated by active forces, described by a continuous-time dynamical model.
2. Longevity is the total duration of mind-body coupling, dominated by active forces and constrained by passive forces, described by a probability-duration model.
3. The two constructs are coupled (shared aging substrates) yet decoupled (independently regulable), with active force as the shared core regulatory node.
4. The model integrates the 14 hallmarks of aging, gene-environment weights, inter-organ communication, and lifestyle choices, with robust empirical grounding and broad applicability.

Future Directions

1. Fit model parameters (weights, thresholds) using large population cohorts
2. Develop wearable/AI real-time computing systems for clinical translation
3. Conduct active force intervention trials to validate health/lifespan benefits
4. Extend to population, ecological, and social health for a unified systems theory

This framework provides a unified mathematical and scientific foundation for transcending materialism, integrating mind-body-support, maximizing healthspan, and minimizing untimely death risk, propelling health science and geriatric medicine into a new paradigm of systems theory, post-materialism, and precision.

Author Contributions

This is a single-author paper; the author contributed to all aspects of the research and writing.

Funding

This research was supported by the Scientific Research Project of Westlake University “Theoretical Research and Demonstration Application of Complex Systems and Deep-Sea Technology (Phase I)” (Grant No. WU2025A006).

Data Availability Statement

No datasets were generated or analyzed during the current study; further inquiries can be directed to the corresponding author.

Acknowledgments

The motivation for this paper arose from a discussion with Prof. Xinge Yu of City University of Hong Kong and Dr. Suming Bian of Westlake University at the Symposium on Advanced Electronics and Sensing Technologies, held at Westlake University on March 29, 2026. During the symposium, I asked how their measured data could be applied to predict health status and lifespan. I am grateful to them for this insightful and constructive discussion. The author thanks Doubao (an AI developed by ByteDance) for its support in literature search and Chinese-to-English translation during the research and manuscript drafting process. The author has reviewed and edited all AI-generated output and takes full responsibility for the content of the publication.

Conflicts of Interest

The author declares no conflicts of interest. The funders had no role in the design of the study; the collection, analysis, or interpretation of the data; the writing of the manuscript; or the decision to publish the results.

REFERENCES

Argentieri, M.A., Amin, N., Nevado-Holgado, A.J., et al. (2024). Integrating the environmental and genetic architectures of aging and mortality. *Nature Medicine*, 31, 1016-1025. <https://doi.org/10.1038/s41591-024-03483-9>

Bian, Z., Wang, L., Fan, R., Sun, J., Yu, L., Xu, M., et al. (2024). Genetic predisposition, modifiable lifestyles, and their joint effects on human lifespan: evidence from multiple cohort studies. *BMJ Evidence-Based Medicine*, 29(4), 255-263. <https://doi.org/10.1136/bmjebm-2023-112583>

- Bianconi, G., et al. (2023). Complex systems in the spotlight: next steps after the 2021 Nobel Prize in Physics. *Journal of Physics: Complexity*, 4, 010201. <https://doi.org/10.1088/2632-072X/ac7f75>
- Cagan, A., Baez-Ortega, A., Brzozowska, N., et al. (2022). Somatic mutation rates scale with lifespan across mammals. *Nature*, 604, 517-524. <https://doi.org/10.1038/s41586-022-04618-z>
- Castellani, B., Gerrits, L. (2021). Map of the complexity sciences. Art and Science Factory, LLC. <https://sacswebsite.blogspot.com/2021/09/q-for-2021-version-of-map-of-complexity.html>
- Cui, W. (2026a). On logical circularity in life sciences: A comparative study based on Darwinian hypothesis and Buddhist hypothesis. *Journal of Scientific Exploration* (in press).
- Cui, W. (2026b). Can Life Be Designed?—An Exploration From the Perspective of the Unified Complex Systems Theory. *Philosophy Study*, 16(1), 58-73. <https://doi.org/10.17265/2159-5313/2026.01.005>
- Cui, W. C. (2026c). Reconstruction of mathematical philosophy and fundamental axiom system based on unified complex system theory. *Philosophy Study*, 16(3), 269-282. <https://doi.org/10.17265/2159-5313/2026.03.008>
- Cui, W. C. (2026d). On the rationality of aether as an ontological candidate in physics. *Philosophy Study*, 16(3), 190-198. <https://doi.org/10.17265/2159-5313/2026.03.002>
- Cui, W., & Pan, L. (2025). A systems theory perspective on science, philosophy, the universe, and human existence. *Journalism and Communication Science Bulletin*, 2(2), 17-28. <https://doi.org/10.48014/jcsb.20250616001>
- Cui, W., Li, R., & Pan, L. (2025). Unified Complex Systems Theory (UCST): Resolving Materialist Dilemmas Through Dualist Ontology and Active Force. *European Journal of Applied Sciences*, 13(05), 517-545. <https://doi.org/10.4236/ejas.2025.135033>
- Garmany, A., Terzic, A. (2025). Healthspan-lifespan gap differs in magnitude and disease contribution across world regions. *Communications Medicine*, 5, 381. <https://doi.org/10.1038/s43856-025-01111-2>
- Guo, J., Han, X., Qin, Y. et al. (2026). Interleukin-10 expressing B lineage cells in visceral adipose tissue protect against aging-related insulin resistance and extend lifespan. *Nat Commun*, 17, 2466. <https://doi.org/10.1038/s41467-026-69371-7>
- López-Otín, C., Kroemer, G. (2025). Hallmarks of aging: Integrating molecular and social determinants. *Geromedicine*, 1, 202507. <https://doi.org/10.70401/Geromedicine.2025.0007>
- Lu, J., Tu, W., Li, R., et al. (2025). Prevalent mesenchymal drift in aging and disease is reversed by partial reprogramming. *Cell*, 188(21), 5895-5911.e17. <https://doi.org/10.1016/j.cell.2025.07.031>
- Molinero, N., Antón-Fernández, A., Hernández, F., Ávila, J., Bartolomé, B., Moreno-Arribas, M.V. (2023). Gut microbiota, an additional hallmark of human aging and neurodegeneration. *Neuroscience*, 518, 141-161. <https://doi.org/10.1016/j.neuroscience.2023.02.014>
- Olshansky, S.J. (2022). From life span to health span: Declaring “victory” in the pursuit of human longevity. *Cold Spring Harbor Perspectives in Medicine*, 12(12), a041480. <https://doi.org/10.1101/cshperspect.a041480>
- Pianese, L., Bordoni, B. (2025). Beyond the biomedical model: A critical review of the approach to chronic pain and the proposal of an integrated functional model. *Cureus*, 17(6), e85717. <https://doi.org/10.7759/cureus.85717>
- Picca, A., Faitg, J., Auwerx, J., Ferrucci, L., D’Amico, D. (2023). Mitophagy in human health, ageing and disease. *Nature Metabolism*, 5(12), 2047-2061. <https://doi.org/10.1038/s42255-023-00930-8>

- Roach, M., McNally, C., & Gordon, M. (2009). *Karmic management: What goes around comes around in your business and your life*. Harmony Books.
- Shenhar, B., Pridham, G., De Oliveira, T. L., Raz, R., Yang, Y., Deelen, J., Hägg, S., Alon, U. (2026). Heritability of intrinsic human life span is about 50% when confounding factors are addressed. *Science*, 391(6784), 504-510. <https://doi.org/10.1126/science.adz1187>
- Sterling, P. (2023). A challenge to neuro-reductionism. *Current Biology*, 33(24), R1266-R1267. <https://doi.org/10.1016/j.cub.2023.11.006>
- Tart, C. T. (2009). *The end of materialism: How evidence of the paranormal is bringing science and spirit together*. New Harbinger Publications; Noetic Books.
- Tokizane, K., Imai, S. I. (2025). Inter-organ communication is a critical machinery to regulate metabolism and aging. *Trends in Endocrinology & Metabolism*, 36(8), 756. <https://doi.org/10.1016/j.tem.2024.11.013>
- Walach, H. (2019). *Beyond a materialist worldview: Towards an expanded science*. Scientific and Medical Network. <https://galileocommission.org/wp-content/uploads/2020/02/Science-Beyond-A-Materialist-World-View-Digital-18.02.20.pdf>
- Wang, G., Song, A., & Wang, Q. A. (2025). Adipose tissue ageing: implications for metabolic health and lifespan. *Nature Reviews Endocrinology*, 21, 623-637. <https://doi.org/10.1038/s41574-025-01142-8>
- Yaku, K., Okabe, K., Nakagawa, T. (2018). NAD metabolism: Implications in aging and longevity. *Ageing Research Reviews*, 47, 1-17. <https://doi.org/10.1016/j.arr.2018.05.006>