

Commentary

Cognitive Hormesis and Somatic Maintenance: A Systems-Based Perspective on Human Aging

Marios Kyriazis¹

1. National Gerontology Centre, Cyprus

Compared to a few decades ago, our environment has undergone a profound transformation. In technologically advanced societies, individuals are increasingly embedded within complex, information-rich digital networks which demand unremitting cognitive interaction. These networks, increasingly encompassing digital Artificial Intelligence (AI) systems, can be viewed as complex adaptive systems in which individuals act as agents contributing to overall system functionality and adaptability.

Within this context, we propose an integrated framework that combines two complementary concepts: the Indispensable Soma Hypothesis and the Law of Requisite Usefulness. The former suggests that positive (hormetic) cognitive engagement may contribute to neuronal maintenance through activation of existing neurobiological adaptive pathways. The latter introduces a systems-level principle, whereby agents which contribute to the overall adaptability and stability of any complex system tend to become more integrated and functionally significant within that system. The aim of this paper is to explore how these concepts may be linked and how these can be applied in a practical way in order to reduce the impact of human aging. Specifically, we examine the hypothesis that individuals who are meaningfully engaged, both cognitively and socially, within our complex, AI-mediated environments may experience indirect biological benefits through mechanisms associated with environmental enrichment, hormesis, soma-to-germline communication, and brain-body signalling. Our hypothesis complements existing models. Cognitive engagement modulates systemic repair pathways and enhances maintenance through hormetic and neurobiological pathways, which may indirectly influence aging and resource allocation, specifically in an increasingly digitalised society.

1. Introduction

A central objective in the biology of aging is to understand as many mechanisms as possible, which drive time-related functional decline, while identifying modifiable factors that may improve health span. Traditional frameworks, including the disposable soma theory ^[1] and antagonistic pleiotropy ^[2], emphasize trade-offs between somatic maintenance and reproduction, largely governed by evolutionary pressures that diminish after reproduction. While these models have provided valuable insights, they do not fully account for the variability observed in human aging ^[3], particularly in the context of modern, technologically enriched environments ^[4]. It is evident that every individual ages differently due to a variety of interconnected intrinsic and extrinsic factors.

Aging is a complex process involving not only biological, but also societal, and technological dimensions. This approach emphasizes that while biological erosion occurs, successful aging is heavily influenced by environmental, social, and psychological factors, requiring personalized support in order to promote a healthier and longer life. This view shifts the focus from purely biomedical decline to a holistic, "time-related dysfunction" model ^[5].

During the past few decades, increasing attention has been directed at the role of environmental and behavioral factors in modulating aging. Concepts such as hormesis and environmental enrichment ^[6] suggest that exposure to mild, adaptive physical, cognitive or social stressors, can activate cellular repair pathways, enhance resilience, and improve useful function ^{[7][8][9]}. In particular, cognitive engagement is emerging as a key determinant of brain health, with evidence linking sustained mental activity to reduced risk of cognitive decline and improved overall well-being ^{[10][11]}. Digital ecosystems involve continual exposure to rapidly changing, meaningful information requiring adaptive responses, which lead to persistent neuronal hormesis and altered evolutionary selection pressures on human maintenance systems.

In this Viewpoint paper we are examining the influence of an increasingly cognitive, digitalised environment, upon certain aspects of the biology of aging, suggesting that aging is modulated not only by genetic and environmental factors, but by the organism's level of integration within complex adaptive high-information systems. Continued engagement with such complex systems (such as a digitalised AI society) may generate hormetic and neurobiological signals that help maintain somatic integrity, thereby

ensuring health span and extending functional lifespan. We introduce the concept that the human informational environment has become an evolutionary driver. Biology has long recognized nutrition, infection, temperature, toxins, social and physical activity as environmental drivers. We are proposing that, in the twenty-first century, AI-mediated and interactive information has become another environmental force acting on human biology.

2. Hormesis and the neuronal stress response

The concept of hormesis has been gaining acceptance in many fields of biological and clinical medicine. Hormesis is a biphasic (U-shaped) biological response where low/moderate exposure to a stressor or toxin leads to beneficial and adaptive effects, while higher doses of the same stressor are harmful, and very low doses are ineffective. The concept operates on the principle that mild, intermittent stress triggers repair mechanisms, thus increasing resistance to future stress ^[12]. This protective mechanism is based on stimulation of cytoprotective pathways, such as the production of antioxidants, molecular chaperones (for instance, heat shock proteins), modulation of autophagy, proteostasis, apoptosis, and DNA repair mechanisms ^[13]. Hormesis helps cells maintain longevity through constant, controlled adaptation. Examples of hormesis include physical exercise, phytochemicals, dietary restriction, and cognitive stimulation ^[14].

Within the framework of hormesis, we propose that mild stress originating from sustained engagement with cognitively-demanding, information-rich environments (such as those encountered in our modern digitally-mediated information ecosystems, and AI settings) may contribute to the maintenance of somatic integrity and resilience during aging by activating the neuronal stress response through neurobiological hormetic, and systemic adaptive pathways. These pathways also include neuroplasticity and neuroendocrine signalling, which collectively enhance cellular repair, resilience, and functional capacity across multiple biological levels. In this context, neuronal activity represents a metabolically and functionally significant component of the soma, capable of influencing organismal homeodynamics through established brain–body communication pathways.

3. The Indispensable Soma Hypothesis

Our Indispensable Soma Hypothesis ^{[15][16]} is mechanistic extension of this framework, and proposes that when a human is deeply integrated into a complex technological or cognitive society, their survival

becomes very relevant to that society. As a result, cellular repair resources may shift from the germline and are re-directed toward the somatic cells (neurons) thus delaying the process of aging. Instead of investing heavily in reproductive cells (as is traditionally dictated by evolutionary biology), the organism may shift several aspects of its biological maintenance and repair resources to the soma (particularly the neurons) to keep the individual functional. The hypothesis assumes that participating in a hyper-connected, information-rich, AI-mediated ecosystem creates unique evolutionary pressures, leading to the suggestion that age-related degeneration can be naturally mitigated through this environmental and cognitive immersion, rather than solely relying on medical or pharmacological interventions ^[16].

Early indications from model organisms show that bidirectional communication exists between germline and somatic tissues, mediated through molecular and epigenetic pathways ^{[17][18]}. While these interactions are well documented in lower organisms ^{[19][20]}, their relevance to human aging remains incompletely understood. We hypothesize that, in humans, sustained neuronal activation as a result of interacting with complex cognitive and AI-based environments may influence these soma-to-germline dynamics ^[16]. Importantly, this hypothesis does not assume a deterministic redistribution of biological resources from germline to soma. Rather, it suggests that behavioral and environmental factors—particularly those involving meaningful cognitive stimulation—can modulate systemic physiology in ways that favor somatic (neuronal) maintenance and delay aspects of age-related functional decline. This framework proposes that aging may be influenced by the degree of an individual's engagement with complex, adaptive digital environments ^[21]. It is plausible that sustained neuronal activation may influence systemic resource allocation through established brain–body signalling networks. Further empirical research is required to clarify the underlying mechanisms and to determine the extent to which these processes contribute to human longevity and health span.

4. Requisite Usefulness

Nested within the Indispensable Soma Hypothesis is the concept of *requisite usefulness* ^[22]. This concept suggests that *'any agents (biological, human, digital) which contribute to system adaptability, tend to occupy positions that increase their own probability of survival within that system.* In complex adaptive systems (such as a human organism, or a human society), participants who enhance the stability and functional integration of such systems, tend to become more connected, stable, and influential within the network. We hypothesize that this systems-level integration may indirectly influence biological aging. Specifically, individuals who are cognitively and meaningfully engaged are more likely to experience sustained

activation of the neurobiological pathways mentioned above, pathways associated with environmental enrichment and hormesis, which may support somatic maintenance and reduce the impact of aging [\[6\]\[13\]\[23\]](#). These pathways include modulation of stress responses, enhancement of neuroplasticity, and downstream effects on immune, metabolic, and repair mechanisms.

For our purposes, 'Usefulness' is "the degree of positive participation of an agent in complex, information-rich adaptive networks". We do not suggest that usefulness itself modifies biology; rather, usefulness increases the probability of repeated exposure to cognitively demanding environments that activate known adaptive repair pathways. A component of a complex system is considered 'useful' when it contributes significantly to the system's ability to create, maintain, and adapt to emerging patterns, usually in an interactive, non-linear manner. Examples include maintaining resilience during disturbances (homeostasis), enhancing learning through feedback loops, and producing emergent, innovative biological solutions.

Importantly, this integration is not proposed to directly alter biological aging through a top-down selection process. Rather, it is likely mediated through indirect pathways. Individuals who are more engaged within their environments tend to exhibit behavioral and lifestyle patterns associated with improved health, including increased cognitive stimulation, social interaction, purpose-driven activity, and adaptive stress exposure. These factors are known to influence physiological systems in ways that support resilience and functional maintenance [\[24\]\[25\]](#). The Law of Requisite Usefulness is not proposed as a physical law but as a systems principle analogous to principles used in cybernetics and complex adaptive systems.

5. Discussion

According to the hypothesis under consideration here, we are suggesting that modern AI-mediated digital environments may represent the most intense and scalable form of meaningful cognitive engagement yet encountered during human evolution. Those individuals who are embracing this new ecological niche, tend to remain biologically functional for longer within that environment. These humans may be able to broadly reduce the impact of their aging and thus survive for longer because they are repeatedly exposed to adaptive stimuli which favour neuronal maintenance [\[26\]](#).

For a significant section of humanity, the current environment is now becoming considerably different compared to previous environments. Our modern environment is no longer a community of interacting

species, but increasingly, a combination of just two principal elements: humans and computers (artificial intelligence/internet). Systems tend to increase both the fitness and the life-span of their individual components if these components provide useful functional feedback to the system.

The cybernetic concept of ‘Selective Reinforcement’ ^{[27][28]} provides additional theoretical support for the above submission. The concept holds that system dynamics tend to favour an agent (or action) if its content of information is of a sufficient and appropriate magnitude. This is a universal theme also encountered in the survival of neurons in the brain. The concept can be extrapolated to humans who act as autonomous creative agents within a technological society. Such creative humans improve the flexibility of the system and provide many degrees of freedom, enabling the system to store more useful information and thus enhance functionality, adaptation and evolvability (i.e. longer survival). Attempts have already been made to elaborate on some possible biological mechanisms which may potentially underpin this increased physical longevity. One such mechanism involves information - sharing processes which facilitate a re-allocation of resources from germline cells to somatic cells. This was briefly mentioned above, and will be discussed in more detail below. In addition, it has been shown that appropriate external stimulation may cause epigenetic reprogramming, where an adult cell (old) can revert to a pluripotent (young) stem cell, and even that this epigenetic information may be heritable ^[29] ^{[30][31]}.

The framework proposed here attempts to bridge biological, behavioral, and systems-level perspectives on aging. At its core lies the idea that sustained cognitive engagement within complex digital, AI-mediated environments can influence organismal physiology through well-established mechanisms, including hormetic stress responses, which modulate immune function, metabolic regulation, and cellular maintenance and repair. Evidence from experimental and observational studies supports several components of this framework. Environmental enrichment has been shown to improve cognitive function, enhance neuroplasticity, and exert systemic effects on immunity, inflammation, and tissue repair ^{[32][33][34][35]}. Similarly, hormetic stimuli—including cognitive challenges—can activate stress-response pathways that promote cellular maintenance and expand the organism’s homeodynamic capacity ^{[13][14]}. While much of the detailed mechanistic evidence derives from model organisms, there is growing support for analogous processes in humans ^{[6][14][36]}.

The interaction between somatic and germline maintenance systems represents an additional area of interest. Sustained neuronal activation may influence organismal homeostasis through indirect signalling pathways, and perhaps, (more speculatively) through direct competition with the germline for

resources. In a study by Wong et al. [37] it was shown that microRNAs which are normally found in the brain, may act on germline mRNA targets, modifying gene expression in the germline.

It has also been suggested [38] that somatic elements may interfere with apoptosis of germline cells, at least in *C.elegans*, where somatic factors (such as CEP-1) were found to modulate apoptosis in gonadal cells. This strengthens the position that the nervous system is able to influence events on the germline. This framework also resonates with broader trends observed in modern societies. As environments become increasingly cognitive and information-driven, there is a shift toward activities that demand continuous mental engagement and adaptation. At the same time, demographic patterns such as increased lifespan and reduced fertility have emerged. While these phenomena are influenced by multiple factors, including socioeconomic conditions, it is worth considering whether long-term changes in cognitive and environmental demands may also contribute to shaping biological aging processes. We know for example, that there is a reciprocal relationship between cognitive function and inflammation, where higher cognitive ability or greater engagement is associated with lower levels of peripheral pro-inflammatory markers (such as C-reactive protein, IL-6, and TNF-alpha) [39].

In summary, we claim that fostering cognitive engagement, with exposure to meaningful information that requires us to act, should protect against long-term age-related decline. The key message here, is to embrace human-computer co-creativity. Human aging evolved under conditions where cognitively demanding environmental challenges were intermittent, local, and relatively slow. Contemporary AI-mediated digital ecosystems expose the brain to an unprecedented volume, speed, complexity, and interconnectedness of meaningful information requiring continual adaptive responses. We hypothesize that this novel informational ecology constitutes a sustained hormetic stimulus capable of enhancing neuronal maintenance, systemic resilience, and perhaps evasion of aging.

6. Critique and Justifications

6.1.

Classic evolutionary theories suggest that selection pressure declines sharply after reproduction. Our hypothesis creates an evolutionary tension, because it implies that continued “usefulness” can maintain somatic investment even post-reproductively. The question here is “Why should evolution maintain the soma for post-reproductive usefulness?” One possible answer is provided by the Evolvable Soma Theory of Aging (ESTA). In recent computational studies by us [40][41], we introduced the ESTA, positing that

aging itself may be a form of evolutionary experimentation. We modelled evolution in such way that gene expression timings could evolve. This evolvable soma scenario outperformed classic models—suggesting organisms may maintain the soma deliberately as a substrate for evolutionary ‘testing’. With age progression, somatic cells express genetically-encoded epigenetic changes—some deleterious, some potentially adaptive—allowing evolution to test innovations in a period characterized by reduced evolutionary pressure and lower risk. This reframes soma not as “disposable,” but as necessary substrate for exploration by evolutionary processes, a reframing aligning with our hypothesis.

ESTA posits that aging is not merely a byproduct of limited evolutionary pressure, but an active, genetically driven process, where cumulative bodily damage results from evolutionary experiments in late life. It suggests that these late-life adaptations, driven by epigenetic mechanisms, are evolutionary attempts to refine species design with lower fitness costs. ESTA simulations suggest that the soma is preserved for evolutionary flexibility, and not simply discarded. Together, these findings converge on a unified insight: the soma cannot be considered as always disposable. When its integrity benefits organismal adaptability, resilience and healthy evolution, then it becomes indispensable.

6.2.

In our approach, we claim that meaningful cognitive stimulation may divert repair resources from germline to neurons. This is speculative at present, although the general concepts behind this remain valid: we know that germline–soma communication exists (mostly shown in worms and flies), hormetic stress improves repair, and that brain activity affects systemic physiology. The idea that “germline repair decreases as neuronal repair increases” describes a fundamental evolutionary trade-off in resource allocation, where organisms prioritize the maintenance of somatic tissues (specifically, the brain) over the long-term integrity of their germline cells. There is a soma-to-germline conflict exemplified by the “expensive germline” hypothesis. This suggests that maintaining high-fidelity DNA repair in both the germline (egg/sperm) and the soma (body cells) is too energy-intensive ^{[42][43]}. In this trade-off, neurons, which are post-mitotic and essential for survival, are protected by robust repair mechanisms. When these resources reach an upper limit, existing capacities could be exhausted ^[44].

While experimental confirmation of our Indispensable Soma Hypothesis is not yet abundant, a number of emerging scientific frameworks resonate with its core idea: that soma and germline are not rigidly separate, and that somatic maintenance can become indispensable under certain conditions. Evidence for a bidirectional germline–soma signalling is becoming more widespread ^[45]. Traditionally, the Disposable

Soma Theory treats soma as expendable relative to germline. However, recent findings show signals from germ cells protecting the soma under DNA damage stress ^[46] and we also know that some neurodevelopmental disorders show ectopic expression of germline genes (such as DNMT3B, G9A/GLP, MECP2, and KDM5C), in the brain ^[47]. This strongly suggests the relationship is reciprocal, supporting the idea that somatic integrity can elevate when germline status declines. This trade-off is considered an evolutionary compromise for longevity and cognitive function over reproductive perfection ^{[45][48][49]}.

A study in *Caenorhabditis elegans* ^[50] confirmed that germline-derived signals (like piRNAs and Hedgehog-like factors) actively inhibit somatic mitochondrial stress responses, implying that the soma's health is being regulated—and even compromised—based on germline status ^[51]. This mutual dependency supports the idea that the soma is not expendable—it's actively integrated in organismal fitness and potentially indispensable under certain conditions, echoing our notion of a conditional Indispensable Soma.

6.3.

There is a problem with overextension from model organisms to humans. Although there exists evidence that germline–soma dynamics are found in model organisms (*C. elegans*, *Drosophila* ^{[17][50]}), there is no sufficient evidence that this is directly transferable to humans. This is a speculative extrapolation. We address this by arguing:

We know that in extreme/stressful environmental conditions such as very low or very high temperatures, or food scarcity, organisms (including humans) tend to give priority to survival instead of reproduction ^{[52][53]}. Repair resources such as ATP, NAD⁺, amino acids, stem cell activity, and endocrine signals, are diverted from germline maintenance, to somatic maintenance. Similarly, the (positive) cognitive stress we encounter in our modern digitalised society could be conceptualised as such an extreme (stressful/hormetic) situation which triggers prioritization of resources towards survival (of the neuron), over reproduction ^{[15][16]}. Neurons have considerable metabolic demands and they are highly dependent on this continuous diversion of maintenance factors to prevent neurodegeneration and maintain peak cognitive function ^[54], cognitive function which is essential in thriving in an environment saturated with digital information that requires action. However, we acknowledge that this area needs a much more solid grounding and research, that is why we present it as a hypothetical perspective of a scientific framework, and not as a proven theory (Table 1).

1	* Older adults engaging daily in meaningful AI-assisted problem solving should show slower cognitive decline than those with equivalent education but passive digital use.
2	* Biomarkers associated with hormetic adaptation (e.g., BDNF, stress-response proteins, inflammatory profiles) should correlate with sustained meaningful cognitive engagement rather than total screen time.
3	* Passive digital consumption ("doomscrolling") should not produce the predicted benefits and may even have the opposite effect.
4	* The quality, novelty, and action-requirement of information should predict outcomes better than the quantity of simple digital exposure.
5	* Future prospective, controlled, graded-dose studies with health and cognition endpoints should show a U-shaped pattern hormesis-like stress-response signature.

Table 1. Falsifiable predictions arising from this framework

6.4.

Our position is that meaningful cognitive engagement within a complex society leads to mild neuronal stress (hormesis) and then to adaptive repair. But is this a justified claim?

A. Overextension of the presence of general hormesis

A relative weakness of our hypothesis is that it does not provide a direct test of cognitive hormesis in human aging. Direct, rigorous human aging trials testing the proposed dose-quality boundaries are not discussed as these are not yet available, although preliminary evidence exists ^{[12][14]}. Our paper is a narrative synthesis which is based on cross-species extrapolation and increases the risk of overgeneralization to humans. In humans, factors such as individual risk profiles, comorbidities, medications, age-related degenerative conditions, and physiological vulnerability matter, and biological plausibility does not always justify universal generalisations.

B. Does digital exposure lead to hormetic (beneficial) stress?

Another potentially vulnerable point of our hypothesis is our argument that, specifically, digital information could cause hormetic (mild but useful) cognitive stress. In fact, digital overload can impair

cognition. Although evidence for cognitive stimulation resulting in neuronal benefit exists, evidence for digital exposure specifically causing hormesis is weaker, but possible ^[55]. Digital hormesis depends on "meaningful" exposure. The key here is that digital exposure should be engaging, new, and requiring action (constructive/creative thinking) rather than mere passive, addictive scrolling. Low-quality exposure to digital information, with superficial, rapidly digested content (low quality scrolling for example), has been linked to "brain rot" ^[56]. Therefore, the digital exposure must *not* be trivial or unchallenging, but meaningful, important, valuable, complex, and requiring a positive effort. This type of digital exposure triggers a mild stress response in neurons ^{[55][57][58][59][60]}, activating molecular mechanisms that improve synaptic plasticity and neuronal health ^[61]. Potential positive outcomes include increased cognitive adaptability, reduced risk of age-related cognitive decline, and improved mental well-being when used as part of an active lifestyle. Similar to physical exercise, this stimulus has a beneficial effect within a specific range, the 'Hormetic Window'. Overexposure to digital content can exceed this threshold, leading to detrimental effects such as anxiety, addiction, and decreased mental performance.

Some preliminary evidence supports the presence of cognitive hormesis in humans ^[62]. Our own research shows that appropriate video games may improve cognitive faculties in older people ^{[63][64]}. In addition, digital cognitive stimulation (DCS) or computerized cognitive training (CCT) are promising interventions that increase brain-derived neurotrophic factor (BDNF) levels, facilitating neuroplasticity, enhanced synaptic connectivity, and improved cognitive performance, particularly in older adults ^[65]. Beneficial digital exposure refers to the strategic and meaningful use of technology. Examples of beneficial digital exposure in humans include the use of interactive educational programs and actively engaging with novel information, constructive use of apps, AI or platforms that encourage creative thought, and movement-based apps or active video games that promote physical activity ^{[66][67][68]}. In order to maximize the benefits of digital hormesis, intentional and moderate engagement is required, often balanced with periods of digital detox ^[64]. When applied to Artificial Intelligence, the framework of cognitive hormesis suggests that manageable, "meaningful" challenges, such as problem-solving with AI (human-computer creativity ^[69]), and other unfamiliar, complex information exposure can act as a 'digital exercise' and enhance brain function ^{[70][71][72][73][74]}.

7. Summary and Conclusion

While our hypothesis awaits robust direct empirical confirmation, it is strongly supported by theoretical simulations, empirical data on germline-soma signalling, and valid extensions of existing aging models, such as activation of proteostasis ^[75], all of which may be contributing to a shift in how we conceptualize the soma: from disposable, to conditionally indispensable. These notions reinforce the idea that somatic integrity must be actively managed to delay systemic drift—moving the soma from failure-prone, to co-regulated with survival and adaptation. Together, these findings converge on a unified insight: the body is not always disposable—it may be indispensable when its integrity benefits the adaptability and resilience of the human society. Our position is that meaningful cognitive engagement within a complex technological society may lead to mild neuronal stress (hormesis) and then to adaptive repair. As a powerful systems-level extension of existing theories, our model proposes that the soma is maintained longer when it remains functionally indispensable within a complex adaptive system, which, in this case, is the modern AI-mediated society. This model aligns with several existing notions such as the Disposable Soma Theory (but with inverted emphasis), antagonistic pleiotropy, hormesis (stress-induced adaptive maintenance), neuroplasticity and cognitive reserve. As a key contribution, we shift aging from passive decline to conditional maintenance based on systemic value. This also aligns with other emerging views on ageing, and it integrates multiple domains such as cellular biology, evolutionary theory, information/complex systems, behaviour and cognition (Figure 1). These proposed organising principles are not simply elegant summaries, but genuinely necessary explanatory structures.

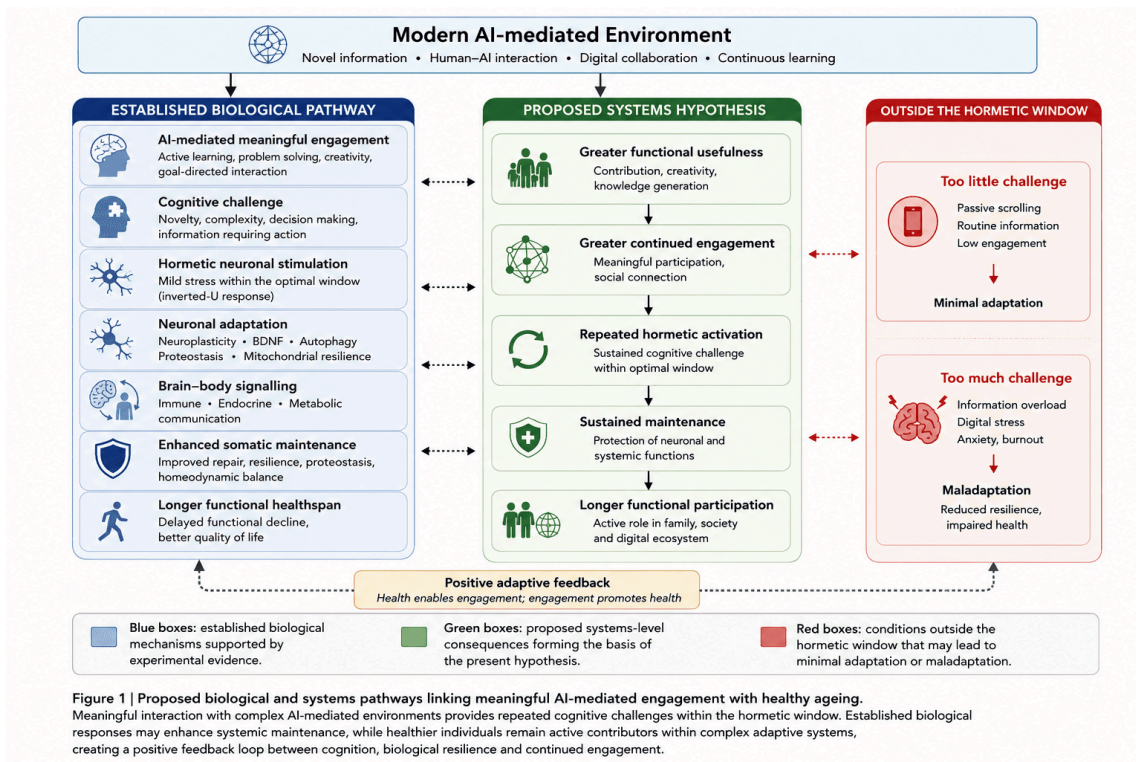


Figure 1.

Several limitations are acknowledged. The hypothesis presented here is integrative and conceptual, and, while it is grounded in established biological principles, direct empirical evidence linking system-level integration to human longevity remains limited, although promising. Longitudinal studies and interdisciplinary approaches will be particularly valuable in testing these ideas (Table 1).

The integration of the Indispensable Soma Hypothesis with the Law of Requisite Usefulness offers a novel perspective on aging, emphasizing the interplay between cognitive engagement, system integration, and biological maintenance. Rather than viewing aging solely as a passive process of decline, this framework suggests that it may be modulated by the extent to which individuals remain actively engaged within digitally complex, adaptive environments. Here, we use concepts such as hormesis, environmental enrichment, neuronal stress response, and soma-to-germline signalling, which are solid and defensible, well supported by research, mechanistically plausible, and clinically relevant. Our perspective is a systems biology extension of hormesis and plasticity, where continued engagement in complex adaptive environments may maintain organismal integrity, and reduce age-related

degeneration. Positive AI-mediated interaction can become a biological driver of healthy aging but only meaningful, adaptive engagement that remains within the hormetic window is beneficial.

The central claim of this paper is not that all these mechanisms have been fully demonstrated, but that together they define a coherent conceptual framework generating experimentally testable predictions. Modern digital environments provide an unprecedented real-world experimental context in which established biological principles may interact in ways not previously encountered during human evolution.

Glossary

- **AI-mediated environment** = a setting where artificial intelligence tools are so deeply embedded that interacting with AI is a constant, unavoidable part of daily operations
- **Indispensable** = functionally difficult to replace within the adaptive dynamics of a complex system
- **Cognitive engagement** = the sustained, active mental effort to understand complex ideas or master challenging tasks, defined by deep processing rather than superficial learning
- **Hormesis** = a biphasic biological response where low doses of a stressor or toxin generate positive, adaptive effects, while high doses of the same stressor are harmful
- **Meaningful information** = ‘information-that-requires-action’, NOT trivial information
- **Meaningful cognitive engagement** = information processing that simultaneously requires novelty, interpretation, decision-making and behavioural or creative response
- **Digitalised environment** = an environment saturated with exposure to digital information
- **Soma** = All living matter of an animal except the reproductive cells. Here, we refer specifically to humans, and primarily to neurons
- **Germline** = Cells that are dedicated to reproduction (sperm and eggs)
- **Agent** = Anyone or anything that has the power to act and produce an effect

Statements and Declarations

Funding

No specific funding was received for this work.

Potential Competing Interests

No potential competing interests to declare.

Author Contributions

- MK: Project administration, Concept development, Supervision, Writing – review & editing.
- SG: Validation, Additions, Writing – review & editing
- AF: Investigation, Resources, Visualization, Writing – review & editing

Use of Generative AI

We used LLM and specifically OpenAI's GPT-5.5 model for general deliberation and for language editing, including grammar, structure and expression, in order to facilitate iterative ideation and improve the clarity and readability of the text. We confirm that we adhered to the JMIR Publications Editorial Policy on the use of generative AI. We reviewed all statements and confirm that these reflect our own views. We independently verified and validated the accuracy of all content and assume total accountability and responsibility for the integrity and originality of the work. All AI-generated outputs have been manually and repeatedly verified by us. No generative AI was used to generate data, produce key scientific findings, draw scientific conclusions, or replace author contributions. The use of AI tools and did not involve manipulation of images or figures beyond standard technical enhancement. We also confirm that all text edited by AI is free from plagiarism and does not infringe copyright or intellectual property rights. All scientific evaluation and final decisions are our own.

References

1. [△]Kirkwood TBL (1977). "Evolution of Ageing." *Nature*. **270**(5635):301–304. doi:[10.1038/270301a0](https://doi.org/10.1038/270301a0).
2. [△]Williams GC (1957). "Pleiotropy, Natural Selection, and the Evolution of Senescence." *Evolution*. **11**(4):398–411. doi:[10.2307/2406060](https://doi.org/10.2307/2406060).
3. [△]Mitchell SE, Simpson M, Coulet L, Speakman JR (2024). "Reproduction Has Immediate Effects on Female Mortality, but No Discernible Lasting Physiological Impacts: A Test of the Disposable Soma Theory." *PNAS*. **121**(42):e2408682121. doi:[10.1073/pnas.2408682121](https://doi.org/10.1073/pnas.2408682121).
4. [△]Kyriazis M (2017). "Editorial: Re-thinking Ageing: A Cross-Disciplinary Perspective (A New Era for Ageing)." *Mech Ageing Dev*. **163**:1. doi:[10.1016/j.mad.2017.04.007](https://doi.org/10.1016/j.mad.2017.04.007).

5. ^aKyriazis M (2020). "Aging as "Time-Related Dysfunction": A Perspective." *Front Med.* 7:371. doi:[10.3389/fmed.2020.00371](https://doi.org/10.3389/fmed.2020.00371).
6. ^{a, b, c}Hanampa-Maquera M, Lourenço RC, Bailey A, Camarini R (2025). "Hormetic Pathways in Environmental Enrichment in Animal Models and Humans." *Prog Brain Res.* 295:189–227. doi:[10.1016/bs.pbr.2025.05.007](https://doi.org/10.1016/bs.pbr.2025.05.007).
7. ^aSun T, Wu H, Cong M, Zhan J, Li F (2020). "Meta-Analytic Evidence for the Anti-Aging Effect of Hormesis on *Caenorhabditis Elegans*." *Aging (Albany NY).* 12:2723–2746. doi:[10.18632/aging.102773](https://doi.org/10.18632/aging.102773).
8. ^aWan Y, Liu J, Mai Y, et al. (2024). "Current Advances and Future Trends of Hormesis in Disease." *npj Aging.* 26. doi:[10.1038/s41514-024-00155-3](https://doi.org/10.1038/s41514-024-00155-3).
9. ^aRattan SIS (2026). "Lifespan Stops at Death, but When Does Healthspan Stop?" *Biogerontology.* 27(2):78. doi:[10.1007/s10522-026-10424-1](https://doi.org/10.1007/s10522-026-10424-1).
10. ^aBasso C, Zanella A, Simoni M, et al. (2026). "Usability and Feasibility of INFORMA Platform for Computerized Cognitive Stimulation in Mild Cognitive Impairment and Early Dementia: An 8-Week Multicenter Study." *Aging Clin Exp Res.* 38(1):99. doi:[10.1007/s40520-026-03346-y](https://doi.org/10.1007/s40520-026-03346-y).
11. ^aLevinson AJ, Ayers S, Clark S, et al. (2026). "Effects of Internet-Based Dementia Risk Reduction Education on Risk and Protective Factor Knowledge, Intentions, and Health Behaviors: Randomized Controlled Trial." *J Med Internet Res.* 28:e79405. doi:[10.2196/79405](https://doi.org/10.2196/79405).
12. ^{a, b}Kawamoto EM, Scavone C, Camarini R (2025). "Physiological Aspects of Hormesis." *Prog Brain Res.* 295:1–7. doi:[10.1016/bs.pbr.2025.05.001](https://doi.org/10.1016/bs.pbr.2025.05.001).
13. ^{a, b, c}Mattson MP, Leak RK (2024). "The Hormesis Principle of Neuroplasticity and Neuroprotection." *Cell Metab.* 36(2):315–337. doi:[10.1016/j.cmet.2023.12.022](https://doi.org/10.1016/j.cmet.2023.12.022).
14. ^{a, b, c, d}Lim VJT, Tabassum NI, Orian JM, Arumugam TV, Weng EF (2025). "Hormesis and Brain Diseases." *Prog Brain Res.* 295:229–258. doi:[10.1016/bs.pbr.2025.05.003](https://doi.org/10.1016/bs.pbr.2025.05.003).
15. ^{a, b}Kyriazis M (2017). "Neurons vs. Germline: A War of Hormetic Tradeoffs." *Curr Aging Sci.* 10(4):242–245. doi:[10.2174/1874609810666170413123547](https://doi.org/10.2174/1874609810666170413123547).
16. ^{a, b, c, d}Kyriazis M, Swas L, Orlova T (2023). "The Impact of Hormesis, Neuronal Stress Response, and Reproduction, upon Clinical Aging: A Narrative Review." *Journal of Clinical Medicine.* 12(16):5433. doi:[10.3390/jcm12165433](https://doi.org/10.3390/jcm12165433).
17. ^{a, b}Ishikawa S, Schumacher B (2025). "The Somatic Impact on Inheritance and the Germline Control of the Soma." *Annu Rev Genet.* 59(1):91–117. doi:[10.1146/annurev-genet-020325-040022](https://doi.org/10.1146/annurev-genet-020325-040022).

18. [△]Lee HJ, Noormohammadi A, Koyuncu S, et al. (2019). "Prostaglandin Signals from Adult Germ Stem Cells Delay Somatic Aging of *Caenorhabditis Elegans*." *Nat Metab.* **1**:790–810. doi:[10.1038/s42255-019-0097-9](https://doi.org/10.1038/s42255-019-0097-9).
19. [△]Conine CC, Rando OJ (2022). "Soma-to-Germline RNA Communication." *Nat Rev Genet.* **23**:73–88. doi:[10.1038/s41576-021-00412-1](https://doi.org/10.1038/s41576-021-00412-1).
20. [△]Gaddy MA, Kuang S, Alfhili MA, Lee MH (2021). "The Soma-Germline Communication: Implications for Somatic and Reproductive Aging." *BMB Rep.* **54**(5):253–259. doi:[10.5483/BMBRep.2021.54.5.198](https://doi.org/10.5483/BMBRep.2021.54.5.198).
21. [△]Dziura SL, Merchant JS, Alkire D, Rashid A, Shariq D, Moraczewski D, Redcay E (2021). "Effects of Social and Emotional Context on Neural Activation and Synchrony During Movie Viewing." *Hum Brain Mapp.* **42**(18):6053–6069. doi:[10.1002/hbm.25669](https://doi.org/10.1002/hbm.25669).
22. [△]Kyriazis M (2015). "Technological Integration and Hyperconnectivity: Tools for Promoting Extreme Human Lifespans." *Complexity.* **20**:15–24. doi:[10.1002/cplx.21626](https://doi.org/10.1002/cplx.21626).
23. [△]Hass S, Liebscher M, Richter A, et al. (2024). "Environmental Enrichment Is Associated with Favorable Memory-Related Functional Brain Activity Patterns in Older Adults." *Front Aging Neurosci.* **16**:1451850. doi:[10.3389/fnagi.2024.1451850](https://doi.org/10.3389/fnagi.2024.1451850).
24. [△]Colditz IG, Campbell DLM, Ingham AB, Lee C (2024). "Review: Environmental Enrichment Builds Functional Capacity and Improves Resilience as an Aspect of Positive Welfare in Production Animals." *Animal.* **18**(6):101173. doi:[10.1016/j.animal.2024.101173](https://doi.org/10.1016/j.animal.2024.101173).
25. [△]Ashokan A, Hegde A, Mitra R (2016). "Short-Term Environmental Enrichment Is Sufficient to Counter Stress-Induced Anxiety and Associated Structural and Molecular Plasticity in Basolateral Amygdala." *Psychoneuroendocrinology.* **69**:189–196. doi:[10.1016/j.psycheneu.2016.04.009](https://doi.org/10.1016/j.psycheneu.2016.04.009).
26. [△]Ihara D, Rasli NR, Katsuyama Y (2025). "How Do Neurons Live Long and Healthy? The Mechanism of Neuronal Genome Integrity." *Front Neurosci.* **19**:1552790. doi:[10.3389/fnins.2025.1552790](https://doi.org/10.3389/fnins.2025.1552790).
27. [△]Prével A, Krebs RM, Kukkonen N, Braem S (2021). "Selective Reinforcement of Conflict Processing in the Stroop Task." *PLoS One.* **16**(7):e0255430. doi:[10.1371/journal.pone.0255430](https://doi.org/10.1371/journal.pone.0255430).
28. [△]Kawato M, Cortese A (2021). "From Internal Models Toward Metacognitive AI." *Biol Cybern.* **115**(5):415–430. doi:[10.1007/s00422-021-00904-7](https://doi.org/10.1007/s00422-021-00904-7).
29. [△]Hochedlinger K, Jaenisch R (2015). "Induced Pluripotency and Epigenetic Reprogramming." *Cold Spring Harb Perspect Biol.* **7**(12):a019448. doi:[10.1101/cshperspect.a019448](https://doi.org/10.1101/cshperspect.a019448).
30. [△]Pereira B, Correia FP, Alves IA, Costa M, Gameiro M, Martins AP, Saraiva JA (2024). "Epigenetic Reprogramming as a Key to Reverse Ageing and Increase Longevity." *Ageing Res Rev.* **95**:102204. doi:[10.1016/j.arr.2024.102204](https://doi.org/10.1016/j.arr.2024.102204).

31. [△]Basu A, Tiwari VK (2021). "Epigenetic Reprogramming of Cell Identity: Lessons from Development for Regenerative Medicine." *Clin Epigenetics*. **13**(1):144. doi:[10.1186/s13148-021-01131-4](https://doi.org/10.1186/s13148-021-01131-4).
32. [△]Figuracion KCF, Lewis FM (2021). "Environmental Enrichment: A Concept Analysis." *Nurs Forum*. **56**(3):703–709. doi:[10.1111/nuf.12565](https://doi.org/10.1111/nuf.12565).
33. [△]Leon M, Woo C (2018). "Environmental Enrichment and Successful Aging." *Front Behav Neurosci*. **12**:155. doi:[10.3389/fnbeh.2018.00155](https://doi.org/10.3389/fnbeh.2018.00155).
34. [△]Baliotti M, Conti F (2022). "Environmental Enrichment and the Aging Brain: Is It Time for Standardization?" *Neurosci Biobehav Rev*. **139**:104728. doi:[10.1016/j.neubiorev.2022.104728](https://doi.org/10.1016/j.neubiorev.2022.104728).
35. [△]Ni MZ, Zhang YM, Li Y, Wu QT, Zhang ZZ, Chen J, Luo BL, Li XW, Chen GH (2022). "Environmental Enrichment Improves Declined Cognition Induced by Prenatal Inflammatory Exposure in Aged CD-1 Mice: Role of N GPF2 and PSD-95." *Front Aging Neurosci*. **14**:1021237. doi:[10.3389/fnagi.2022.1021237](https://doi.org/10.3389/fnagi.2022.1021237).
36. [△]Seider TR, Fieo RA, O'Shea A, Porges EC, Woods AJ, Cohen RA (2016). "Cognitively Engaging Activity Is Associated with Greater Cortical and Subcortical Volumes." *Front Aging Neurosci*. **8**:94. doi:[10.3389/fnagi.2016.00094](https://doi.org/10.3389/fnagi.2016.00094).
37. [△]Wong C, Jurczak EM, Roy R (2024). "Neuronal Exosomes Transport an miRISC Cargo to Preserve Stem Cell Integrity During Energy Stress." *Cell Rep*. **43**(10):114851. doi:[10.1016/j.celrep.2024.114851](https://doi.org/10.1016/j.celrep.2024.114851).
38. [△]Eroglu M, Derry W (2016). "Your Neighbours Matter – Non-Autonomous Control of Apoptosis in Development and Disease." *Cell Death Differ*. **23**:1110–1118. doi:[10.1038/cdd.2016.41](https://doi.org/10.1038/cdd.2016.41).
39. [△]Slaney C, Sallis HM, Jones HJ, Dardani C, et al. (2023). "Association Between Inflammation and Cognition: Triangulation of Evidence Using a Population-Based Cohort and Mendelian Randomization Analyses." *Bra in Behav Immun*. **110**:30–42. doi:[10.1016/j.bbi.2023.02.010](https://doi.org/10.1016/j.bbi.2023.02.010).
40. [△]Fontana A, Kyriazis M (2024). "How Evolution Makes Us Age: Introducing the Evolvable Soma Theory of Ageing." *Biosystems*. **243**:105271. doi:[10.1016/j.biosystems.2024.105271](https://doi.org/10.1016/j.biosystems.2024.105271).
41. [△]Fontana A, Kyriazis M (2025). "Evolvable Soma Theory of Ageing: Insights from Computer Simulations." *Biogerontology*. **26**(2):87. doi:[10.1007/s10522-025-10224-z](https://doi.org/10.1007/s10522-025-10224-z).
42. [△]Chen HY, Jolly C, Bublys K, Marcu D, Immler S (2020). "Trade-off Between Somatic and Germline Repair in a Vertebrate Supports the Expensive Germ Line Hypothesis." *Proc Natl Acad Sci U S A*. **117**(16):8973–8979. doi:[10.1073/pnas.1918205117](https://doi.org/10.1073/pnas.1918205117).
43. [△]Lind MI, Mautz BS, Carlsson H, Hinas A, Gudmunds E, Maklakov AA (2024). "Sex-Specific Growth and Life span Effects of Germline Removal in the Dioecious Nematode *Caenorhabditis Remanei*." *Aging Cell*. **23**:e14290. doi:[10.1111/acer.14290](https://doi.org/10.1111/acer.14290).

44. [△]Maklakov AA, Immler S (2016). "The Expensive Germline and the Evolution of Ageing." *Current Biology*. 26(13):R577–R586. doi:[10.1016/j.cub.2016.04.012](https://doi.org/10.1016/j.cub.2016.04.012).
45. [△][△]Monaghan P, Metcalfe NB (2019). "The Deteriorating Soma and the Indispensable Germline: Gamete Senescence and Offspring Fitness." *Proc Biol Sci*. 286(1917):20192187. doi:[10.1098/rspb.2019.2187](https://doi.org/10.1098/rspb.2019.2187).
46. [△]Teulière J, Bhattacharya D, Baptiste E (2020). "Ancestral Germen/Soma Distinction in Microbes: Expanding the Disposable Soma Theory of Aging to All Unicellular Lineages." *Ageing Res Rev*. 60:101064. doi:[10.1016/j.arr.2020.101064](https://doi.org/10.1016/j.arr.2020.101064).
47. [△]Bonefas KM, Iwase S (2022). "Soma-to-Germline Transformation in Chromatin-Linked Neurodevelopmental Disorders?" *FEBS J*. 289(8):2301–2317. doi:[10.1111/febs.16196](https://doi.org/10.1111/febs.16196).
48. [△]University of California – Riverside (2020). "Neurons Are Genetically Programmed to Have Long Lives." *ScienceDaily*. www.sciencedaily.com/releases/2020/07/200724141020.htm.
49. [△]Lin L, Zhang M, Stoilov P (2020). "Developmental Attenuation of Neuronal Apoptosis by Neural-Specific Splicing of Bak1 Microexon." *Neuron*. 107:1180–1196.e8.
50. [△][△]Zhou L, Jiang L, Li L, et al. (2024). "A Germline-to-Soma Signal Triggers an Age-Related Decline of Mitochondrial Stress Response." *Nat Commun*. 15:8723. doi:[10.1038/s41467-024-53064-0](https://doi.org/10.1038/s41467-024-53064-0).
51. [△]Moses E, Atlan T, Sun X, et al. (2024). "The Killifish Germline Regulates Longevity and Somatic Repair in a Sex-Specific Manner." *Nat Aging*. 4:791–813. doi:[10.1038/s43587-024-00632-0](https://doi.org/10.1038/s43587-024-00632-0).
52. [△]Marzban G, Tesei D (2025). "The Extremophiles: Adaptation Mechanisms and Biotechnological Applications." *Biology (Basel)*. 14(4):412. doi:[10.3390/biology14040412](https://doi.org/10.3390/biology14040412).
53. [△]Bókonyi V, Lendvai ÁZ, Liker A, Angelier F, Wingfield JC, Chastel O (2009). "Stress Response and the Value of Reproduction: Are Birds Prudent Parents?" *The American Naturalist*. 173(5):589–598. doi:[10.1086/597610](https://doi.org/10.1086/597610).
54. [△]Gluck L, Gerstein B, Kaunzner UW (2025). "Repair Mechanisms of the Central Nervous System: From Axon Sprouting to Remyelination." *Neurotherapeutics*. 22(4):e00583. doi:[10.1016/j.neurot.2025.e00583](https://doi.org/10.1016/j.neurot.2025.e00583).
55. [△][△]Small GW, Lee J, Kaufman A, Jalil J, Siddarth P, Gaddipati H, Moody TD, Bookheimer SY (2020). "Brain Health Consequences of Digital Technology Use." *Dialogues Clin Neurosci*. 22(2):179–187. doi:[10.31887/DCNS.2020.22.2/gsmall](https://doi.org/10.31887/DCNS.2020.22.2/gsmall).
56. [△]Yousef AMF, Alshamy A, Tlili A, Metwally AHS (2025). "Demystifying the New Dilemma of Brain Rot in the Digital Era: A Review." *Brain Sci*. 15(3):283. doi:[10.3390/brainsci15030283](https://doi.org/10.3390/brainsci15030283).
57. [△]Korte M (2020). "The Impact of the Digital Revolution on Human Brain and Behavior: Where Do We Stand?" *Dialogues Clin Neurosci*. 22(2):101–111. doi:[10.31887/DCNS.2020.22.2/mkorte](https://doi.org/10.31887/DCNS.2020.22.2/mkorte).

58. ^ΔLeist AK (2013). "Social Media Use of Older Adults – A Mini-Review." *Gerontology*. 59(4):378–384. doi:[10.1159/000346818](https://doi.org/10.1159/000346818).
59. ^ΔSilchenko K (2025). "Technology Dualities and Paradoxes in Digital Well-Being: Analysis of Discourses of Digital Well-Being Market Offerings." *Journal of Macromarketing*. 45(4). doi:[10.1177/02761467251356660](https://doi.org/10.1177/02761467251356660).
60. ^ΔCorreia de Barros E (2024). "Understanding the Influence of Digital Technology on Human Cognitive Functions: A Narrative Review." *IBRO Neuroscience Reports*. 17:415–422. doi:[10.1016/j.ibneur.2024.11.006](https://doi.org/10.1016/j.ibneur.2024.11.006).
61. ^ΔMcLaren-Gradinaru M, Burles F, Protzner AB, Iaria G (2023). "The Cognitive Effects of Playing Video Game s with a Navigational Component." *Telemat Inform Rep*. 9:100043. doi:[10.1016/j.teler.2023.100043](https://doi.org/10.1016/j.teler.2023.100043).
62. ^ΔBenge JF, Scullin MK (2015). "A Meta-Analysis of Technology Use and Cognitive Aging." *Nat Hum Behav*. 9: 1405–1419. doi:[10.1038/s41562-025-02159-9](https://doi.org/10.1038/s41562-025-02159-9).
63. ^ΔKyriazis M, Kiourti E (2018). "Video Games and Other Online Activities May Improve Health in Ageing." *Front Med (Lausanne)*. 5:8. doi:[10.3389/fmed.2018.00008](https://doi.org/10.3389/fmed.2018.00008).
64. ^Δ^ΔKyriazis M, Kiourti E (2019). "Chapter 25 - Interactions with Technology as a Cognitive, Healthy Aging Hormetic Stimulus." In: Rattan SIS, Kyriazis M, editors. *The Science of Hormesis in Health and Longevity*. Academic Press. 285–292. ISBN [9780128142530](https://doi.org/10.1016/B978-0-12-814253-0.00025-5). doi:[10.1016/B978-0-12-814253-0.00025-5](https://doi.org/10.1016/B978-0-12-814253-0.00025-5).
65. ^ΔNicastri CM, McFeeley BM, Simon SS, et al. (2022). "BDNF Mediates Improvement in Cognitive Performance After Computerized Cognitive Training in Healthy Older Adults." *Alzheimer's Dement*. 8:e12337. doi:[10.1002/trc2.12337](https://doi.org/10.1002/trc2.12337).
66. ^ΔLee J, Žarnić Ž (2024). "The Impact of Digital Technologies on Well-Being: Main Insights from the Literature." *OECD Papers on Well-being and Inequalities*. No. 29, OECD Publishing, Paris. doi:[10.1787/cb173652-en](https://doi.org/10.1787/cb173652-en).
67. ^ΔHaddock A, Ward N, Yu R, O'Dea N (2022). "Positive Effects of Digital Technology Use by Adolescents: A Scoping Review of the Literature." *Int J Environ Res Public Health*. 19(21):14009. doi:[10.3390/ijerph192114009](https://doi.org/10.3390/ijerph192114009).
68. ^ΔBolch MB, Moore RM, Robertson GC, Scafe MJ, Milkovich LM (2025). "Screens Are Not the Enemy: Recommendations for Developing Healthy Digital Habits in Youth." *Mo Med*. 122(4):297–303. PMID [40787030](https://pubmed.ncbi.nlm.nih.gov/40787030/).
69. ^ΔGeroimenko V (2025). "Generative AI: From Human–Computer Interaction to Human–Computer Creativity." In: Geroimenko V, editor. *Human–Computer Creativity*. Springer Series on Cultural Computing. Springer, Cham. doi:[10.1007/978-3-031-86551-0_1](https://doi.org/10.1007/978-3-031-86551-0_1).
70. ^ΔRossi S, Fraccaro V, Manzotti R (2026). "The Brain Side of Human-AI Interactions in the Long-Term: The “3 R Principle”." *npj Artif Intell*. 2:15. doi:[10.1038/s44387-025-00063-1](https://doi.org/10.1038/s44387-025-00063-1).
71. ^ΔWesterbeek H (2026). "How AI Is Rewiring the Human Brain: The Generational Transformation of Cognition and Knowing." *AI & Soc*. 41:5327–5337. doi:[10.1007/s00146-026-02912-2](https://doi.org/10.1007/s00146-026-02912-2).

72. [△]Chen Y, Wang Y, Wüstenberg T, Kizilcec RF, Fan Y, Li Y, Lu B, Yuan M, Zhang J, Zhang Z, Geldsetzer P, Chen S, Bärnighausen T (2025). "Effects of Generative Artificial Intelligence on Cognitive Effort and Task Performance: Study Protocol for a Randomized Controlled Experiment Among College Students." *Trials*. 26(1):244. doi:[10.1186/s13063-025-08950-3](https://doi.org/10.1186/s13063-025-08950-3).
73. [△]Luan YL, Kim YJ, Zhou J (2025). "Augmented Learning for Joint Creativity in Human-GenAI Co-Creation." *Information Systems Research* (online). doi:[10.1287/isre.2024.0984](https://doi.org/10.1287/isre.2024.0984).
74. [△]Dai I, van Swol LM (2026). "Who Performs Better? Exploring the Role of Generative AI in Creativity and Brainstorming." *Computers in Human Behavior: Artificial Humans*. 8:100302. doi:[10.1016/j.chbah.2026.100302](https://doi.org/10.1016/j.chbah.2026.100302).
75. [△]Mladenovic Djordjevic AN, Kapetanou M, Loncarevic-Vasiljkovic N, Todorovic S, Athanasopoulou S, Jovic M, Prvulovic M, Taoufik E, Matsas R, Kanazir S, Gonos ES (2021). "Pharmacological Intervention in a Transgenic Mouse Model Improves Alzheimer's-Associated Pathological Phenotype: Involvement of Proteasome Activation." *Free Radical Biology and Medicine*. 162:88–103. doi:[10.1016/j.freeradbiomed.2020.11.038](https://doi.org/10.1016/j.freeradbiomed.2020.11.038).

Declarations

Funding: No specific funding was received for this work.

Potential competing interests: No potential competing interests to declare.