The Science and Practice of Slow Aging: A Comprehensive Report on Modulating the Human Longevity Trajectory

I. The Dawn of Aging Science: From Inevitability to Malleability

1.1. Defining the 'Slow Aging' Paradigm

'Slow aging' signifies a scientific approach that intentionally aims to decelerate the biological aging process, moving beyond simple weight loss or cosmetic enhancements.[1] It stands in contrast to the natural aging process that occurs with the passage of chronological time. The ultimate goal is not merely to extend lifespan but to maintain a high level of health and function until the very end of life.

At the core of this paradigm lies the distinction between **Chronological Age** and **Biological Age**. Chronological age is the time that has passed since birth, an irreversible and absolute value. In contrast, biological age is an indicator of the functional state of our body's cells and tissues, which is variable and dependent on lifestyle, environment, and genetic factors. The fundamental premise of slow aging is that while we cannot change our chronological age, our biological age is well within our control to manage.[2]

The opposite concept is **'Accelerated Aging,'** a state where biological age surpasses chronological age due to improper lifestyle habits or environmental factors.[2] Accelerated aging is a direct cause of the early onset of chronic diseases and functional decline. Slow aging strategies are precisely aimed at preventing this acceleration and slowing its pace. In other words, while aging is an inevitable process that begins at the moment of conception [1], the 'speed' at which it progresses is recognized as a domain that can be controlled by individual effort, marking the starting point of the slow aging philosophy.

1.2. The Concepts of Healthspan, Lifespan, and 'Intrinsic Capacity'

The primary goal of slow aging is not simply to live longer, i.e., the extension of **Lifespan**. More importantly, it is to maximize **Healthspan**—the period of life spent in good health, free from disease or disability—and to minimize the duration of illness.[4] Globally, while life expectancy is increasing, the gap with healthspan is also widening, meaning that the period of old age spent in poor health is growing longer, a significant challenge for modern society.[4]

In this context, the World Health Organization (WHO) emphasizes the concept of **'Intrinsic Capacity'**.[5] Intrinsic capacity is a holistic concept that encompasses an individual's physical and mental abilities. It goes beyond visible indicators like the presence of disease, blood pressure, or exercise time to include a multifaceted state of health covering Mobility, Mentation, Medical issues, and What Matters to the individual.[5] Slow aging aims to actively manage and develop this intrinsic capacity to enable independent living and enhance quality of life in old age.

Aging is the result of a lifetime of accumulation, and when this accumulation becomes severe enough to affect daily life, it leads to a state of **Frailty**.[1] Frailty is a condition where physical function has declined to the point that basic activities like going to the hospital or grocery shopping alone become difficult, and in severe cases, can lead to a bedridden state.[1] Slow aging, through the systematic management of intrinsic capacity, is the most proactive and preventive medical strategy to delay the onset of frailty and extend healthspan.

II. The Molecular Blueprint of Aging: The 12 Hallmarks

2.1. The Foundational Framework: The Original Nine Hallmarks of Aging

A landmark 2013 study that shifted the paradigm of aging research defined nine interconnected biological mechanisms that cause aging as the 'Hallmarks of Aging'.[7] This framework clarified that aging is not the result of a single cause but a complex process of accumulating damage at the cellular and molecular levels. The mechanisms of each hallmark are as follows:

- **Genomic Instability:** The cell's blueprint, DNA, is constantly damaged by both endogenous (e.g., DNA replication errors, reactive oxygen species) and exogenous (e.g., UV radiation, chemicals) factors. With age, the efficiency of the mechanisms that repair this damage declines, leading to the accumulation of mutations, which causes cellular dysfunction and cancer.[7]
- **Telomere Attrition:** Telomeres are protective caps at the ends of chromosomes. Each time a cell divides, the telomeres shorten slightly. When they reach a critical length, the cell can no longer divide and enters a state of 'replicative senescence'.[7]
- **Epigenetic Alterations:** These are changes in chemical tags, such as DNA methylation or histone modifications, that alter gene expression patterns without changing the DNA sequence itself. This leads to the inappropriate activation or suppression of certain genes during the aging process.[7]
- **Loss of Proteostasis:** The systems responsible for maintaining the proper folding of proteins and removing damaged ones (e.g., chaperones, proteasomes, autophagy) become less effective. This results in the accumulation of damaged or misfolded proteins, which can be toxic and cause degenerative diseases like Alzheimer's and Parkinson's.[7]
- **Deregulated Nutrient-Sensing:** Key signaling pathways that regulate cell growth and metabolism, such as Insulin/IGF-1, mTOR, AMPK, and sirtuins, become dysregulated with age. These pathways, essential for growth in youth, can become overactive in old age, causing cells to expend energy on unnecessary growth instead of repair and maintenance.[7]
- **Mitochondrial Dysfunction:** The efficiency of mitochondria, the cell's 'powerhouses,' declines, leading to reduced energy (ATP) production and increased production of harmful reactive oxygen species (ROS). This creates a vicious cycle that exacerbates oxidative damage to other cellular components, accelerating aging.[7]
- **Cellular Senescence:** Damaged or end-of-life cells do not self-destruct but accumulate in the body like 'zombie cells.' These senescent cells stop dividing but continue to secrete inflammatory substances (SASP, Senescence-Associated Secretory Phenotype), causing inflammation in surrounding tissues and promoting aging.[7]
- **Stem Cell Exhaustion:** The number or function of adult stem cells, which are responsible for regenerating and repairing tissues, decreases. This leads to a decline in wound healing ability and an overall reduction in the regenerative potential of tissues, such as muscle loss and weakened immunity.[7]
- **Altered Intercellular Communication:** Problems arise in the signaling systems between cells, leading to a chronic inflammatory state known as 'inflammaging.' This is a result of the combined effects of SASP secretion from senescent cells, declining immune function, and other factors, which promotes a systemic aging process.[7]

2.2. An Expanded Worldview: The 2023 Update to 12 Hallmarks

In 2023, reflecting the research advancements of the past decade, the hallmarks of aging were expanded to twelve.[13] This update signifies that the understanding of aging has broadened from an issue within cells to one involving systemic systems and symbiotic ecosystems.

- **Disabled Macroautophagy:** Autophagy, previously part of 'Loss of Proteostasis,' was elevated to an independent hallmark. This emphasizes the importance of the cellular recycling system that cleans up 'waste' like damaged mitochondria and protein aggregates. The decline in autophagy function has been re-evaluated as a key driver of aging.[13]
- **Chronic Inflammation:** 'Inflammaging,' previously considered a consequence of 'Altered Intercellular Communication,' is now recognized as a central cause of aging. Chronic inflammation is the common denominator underlying almost all age-related diseases and is a key driver that exacerbates other hallmarks.[13]
- **Dysbiosis:** The inclusion of gut microbiome imbalance as a hallmark is one of the most revolutionary changes. It officially acknowledges that aging is not just a process occurring within our bodies but is significantly influenced by our interaction with the microorganisms that live in symbiosis with us.[13] This directly connects to the 'Microbiome' component of the 5M model proposed by Dr. Park Min-soo.[16]

2.3. Interconnectivity and Systemic Collapse

The 12 hallmarks of aging do not operate in isolation but form a complex, interconnected network that creates a synergistic effect.[9] For example, mitochondrial dysfunction (hallmark #7) increases the production of reactive oxygen species, which in turn exacerbates genomic instability (hallmark #1). This damaged DNA can trigger cellular senescence (hallmark #8), and senescent cells secrete inflammatory substances that worsen chronic inflammation (hallmark #11). In this way, one form of damage triggers a cascade of others, leading to a systemic collapse.

The expansion from nine to twelve hallmarks demonstrates a fundamental shift in the perspective on aging. The initial framework focused primarily on issues within the cell (DNA damage, protein denaturation) or between adjacent cells, viewing aging as a failure at the individual cell level. However, the 2023 additions of chronic inflammation and dysbiosis are inherently different. Chronic inflammation is a systemic process involving the entire immune system, while dysbiosis is an issue of an external ecosystem interacting with its host.

This expansion signifies that the scientific community is beginning to recognize aging not just as the breakdown of individual cells, but as a failure of macroscopic regulatory networks like the immune system and symbiotic relationships like the microbiome. This provides a powerful explanation for why holistic lifestyle interventions such as diet, exercise, and stress management are so effective. These interventions do not target a single hallmark but regulate the systemic environment itself (e.g., inflammation levels, gut health), which in turn affects all other hallmarks. This naturally links the abstract scientific principles of this section to the practical guidelines in Section IV.

Table 1: The 12 Hallmarks of Aging: Mechanisms and Modulating Factors
Hallmark
1. Genomic Instability
2. Telomere Attrition
3. Epigenetic Alterations
4. Loss of Proteostasis
5. Disabled Macroautophagy
6. Deregulated Nutrient-Sensing
7. Mitochondrial Dysfunction
8. Cellular Senescence
9. Stem Cell Exhaustion
10. Altered Intercellular Communication
11. Chronic Inflammation
12. Dysbiosis

III. The Frontier of Longevity Science: Can Biological Age Be Reversed?

3.1. Cellular Reprogramming: Rewinding the Epigenetic Clock

The most groundbreaking research suggesting that aging may be reversible comes from the field of **cellular rejuvenation therapy**. Notably, the Salk Institute in the US has successfully reverted aged cells to a younger state using four transcription factors known as the **Yamanaka factors** (Oct4, Sox2, Klf4, c-Myc).[17]

The key to this technology is not to completely revert cells to an undifferentiated embryonic stem cell state, but to induce **'partial reprogramming'** by transiently expressing the Yamanaka factors, thereby maintaining the cell's identity.[19] This process targets 'epigenetic alterations,' one of the hallmarks of aging, by resetting the epigenetic marks of aged cells to the patterns of younger cells.[19]

The results from animal model studies were astonishing. The lifespan of mice with premature aging diseases was extended, and when the therapy was applied long-term to healthy middle-aged and elderly mice, clear rejuvenation effects were observed in various tissues. The epigenetic patterns of kidney and skin cells became similar to those of young mice, and the regenerative capacity of damaged muscle and liver tissue improved. Notably, skin wounds healed faster with less scarring, and metabolic substances in the blood did not show age-related changes.[19]

One of the most significant findings was its safety. In the group of mice that received long-term administration of Yamanaka factors, there was no increase in cancer incidence or other adverse health effects.[19] This suggests that cellular reprogramming technology could potentially be a safe anti-aging therapy. However, it will take considerable time before this technology can be applied to humans, and for now, it is considered a promising but distant future therapeutic possibility.[23]

The most profound implication of cellular reprogramming research is that it fundamentally challenges the conventional view of aging. In the past, aging was considered an irreversible accumulation of 'hardware' damage, like rust on a car. The prevailing belief was that once DNA was altered, it was difficult to repair systemically. However, the Salk Institute's research has shown that it is possible to reset the 'software'—the epigenome that reads the DNA hardware—to a youthful state using Yamanaka factors.[19] This means that cells can become functionally younger without changing their DNA sequence or unique identity.

This implies that a significant portion of the aging phenomenon is not due to permanent hardware damage but to a 'drift' in the control software. If aging is a software problem, then in theory, it is possible to 'debug' or 'reboot' it. This conceptual shift has injected tremendous vitality into the field of aging research and has paved the way for the development of future anti-aging therapies that specifically target the epigenome as a biological target. As a result, aging is gradually moving from the realm of science fiction into the realm of scientific possibility.

3.2. New Horizons in Geroscience and Pharmacology

In addition to cellular reprogramming, various studies targeting the fundamental causes of aging are actively underway, holding promise as future tools for slow aging.

- **NAD+ Precursors:** Substances like nicotinamide riboside (NR) and nicotinamide mononucleotide (NMN) increase cellular levels of NAD+. NAD+ is a coenzyme essential for DNA repair and mitochondrial function, which declines with age. Supplementing NAD+ levels can enhance the cell's maintenance and energy production capabilities.[10]
- **Senolytics:** These are drugs that selectively eliminate senescent cells. A combination of Dasatinib and Quercetin is a prime example, which has been shown in animal studies to reduce chronic inflammation and improve tissue function by clearing senescent cells.[24]
- **mTOR Inhibitors:** Drugs like Rapamycin inhibit the nutrient-sensing pathway mTOR, mimicking the effects of caloric restriction. By suppressing cell growth signals and activating maintenance pathways like autophagy, they are known to extend lifespan.[10]
- **Metformin:** Originally a diabetes medication, Metformin has been found to have broad anti-aging effects and is currently undergoing large-scale clinical trials. It positively influences several aging pathways by activating AMPK and suppressing inflammation.[24]

3.3. Expert Consensus and the Future of Longevity

Currently, scientific experts estimate the maximum human lifespan to be between 115 and 150 years.[23] While a breakthrough in lifespan extension is still considered a 'moonshot' challenge, there is a consensus that significantly extending

healthspan within this century is an achievable goal.[5] As specific methods to slow the biological process of aging are being discovered, aging is transitioning from an inevitable fate to a manageable biological process.

IV. The Pillars of Slow Aging: Evidence-Based Lifestyle Interventions

4.1. Pillar 1: Nutritional Strategies for Cellular Health

4.1.1. Caloric Restriction and Intermittent Fasting

Regulating nutritional intake is one of the most powerful ways to intervene in the speed of aging. In particular, Caloric Restriction (CR) and Intermittent Fasting (IF) directly target the 'deregulated nutrient-sensing' hallmark of aging.

The core mechanism of these strategies lies in modulating key nutrient-sensing pathways within the cell. When food intake is reduced, the **mTOR** signal, which promotes cell growth and proliferation, is downregulated. Conversely, **AMPK** and **Sirtuins**, which are activated in a state of energy deficit and are responsible for cellular repair and maintenance, are upregulated.[7] This shift in signaling is like issuing a command to the cells to activate 'survival and repair mode.'

As a result, the cellular cleaning process known as **autophagy** becomes more active, efficiently removing damaged proteins and mitochondria, and mitochondrial function also improves.[29] Human studies like CALERIE have suggested that long-term caloric restriction can improve cardiovascular metabolic indicators and potentially extend lifespan by 1 to 5 years.[31]

However, maintaining strict caloric restriction for an extended period is practically very difficult. Furthermore, especially in the elderly, there is a risk of side effects such as loss of muscle mass and bone density.[31] For these reasons, intermittent fasting, which allows eating only during specific hours, has recently gained attention as a more practical and sustainable alternative.[32]

4.1.2. Phytonutrients as Modulators of Aging

Phytonutrients, such as **Polyphenols**, which are abundant in plants, were once considered mere antioxidants. However, recent studies have revealed that they have drug-like effects, directly acting on several hallmarks of aging.[25]

The mechanism of action of polyphenols goes far beyond simple free radical scavenging. Resveratrol in red wine, quercetin in onion peels, and EGCG in green tea modulate aging through the following complex mechanisms:

- **Epigenetic Regulation:** They directly influence the activity of epigenetic regulatory enzymes like DNA methyltransferases (DNMTs) and histone deacetylases (HDACs), thereby regulating gene expression.[35]
- **Sirtuin Activation:** They directly activate sirtuins, known as longevity genes, producing effects similar to caloric restriction.[35]
- **Promotion of Cellular Cleanup:** They induce autophagy and mitophagy (selective removal of mitochondria), enhancing intracellular quality control.[35]
- **Inflammation Suppression:** They inhibit key inflammatory signaling pathways like NF-κB, alleviating chronic inflammation.[35]
- **Senolytic Action:** Some polyphenols exhibit a senolytic effect, removing senescent cells and slowing tissue aging.[25]

4.1.3. Synthesis: The Blue Zone Diet as a Practical Model

The diet of people in the world's longevity hotspots, the **Blue Zones**, is an excellent example of how the scientific principles discussed above are implemented in real life.[38] Their diet is not about obsessing over specific nutrients but follows a pattern that naturally aligns with the principles of slow aging.

The core of the Blue Zone diet is a meal plan centered on unprocessed plant-based foods. In particular, the consumption of **legumes (lentils, chickpeas, etc.), whole grains (oats, barley, etc.), nuts, and a variety of colorful vegetables** is consistently high.[38] This type of diet is very rich in polyphenols and fiber, which directly contributes to improving dysbiosis and suppressing chronic inflammation. Additionally, it is generally low in calorie density, naturally aligning with the principle of caloric restriction. Rather than viewing specific foods as 'miracle foods' [1], it is important to practice the scientific principles of slow aging in daily life through such an overall dietary pattern.

4.2. Pillar 2: Movement as Medicine, Exercise and Cellular Rejuvenation

Exercise is more than just a calorie-burning activity; it is a powerful signaling activity that directly counters several hallmarks of aging at the cellular level.[39] Regular exercise reduces genomic instability, improves proteostasis, and enhances mitochondrial function, exerting a comprehensive anti-aging effect.

4.2.1. Comparative Analysis by Exercise Type

Different types of exercise target different aspects of cellular aging, so a complementary approach is necessary.

- **Aerobic Exercise (AE):** Consistent aerobic exercise shows the most consistent effect in increasing **telomerase activity (TA)**, the enzyme that repairs telomeres.[42] This is a key mechanism that directly prevents telomere attrition. It is also essential for improving maximal oxygen uptake (VO2max), a key predictor of longevity.[43]
- **High-Intensity Interval Training (HIIT):** Several meta-analyses suggest that HIIT may be particularly effective in maintaining or increasing **telomere length (TL)**.[44] More importantly, a study from the Mayo Clinic showed that HIIT was superior to other exercise modalities in
  reversing the decline in mitochondrial function and **enhancing the ability to synthesize new proteins** in older adults.[47] HIIT imposes strong stress on cells for a short period, thereby powerfully activating autophagy, mitophagy, and anti-inflammatory pathways.[49]
- **Resistance Exercise (RE):** While the direct impact of resistance exercise on telomeres is not yet clear [42], it is unparalleled in building and maintaining muscle mass. This is the most effective way to directly counter age-related sarcopenia. Since muscle loss is a representative phenotype of stem cell exhaustion and a key element of frailty, strength training is indispensable for maintaining structural integrity and improving insulin sensitivity.[39]

The optimal exercise plan for slow aging is not to stick to a single type of exercise but to create a synergistic combination that complements the targeting of different hallmarks of aging. Research data show that aerobic exercise specializes in activating telomerase, HIIT in restoring mitochondrial and protein synthesis, and resistance exercise in preserving muscle mass.[39] Therefore, the question 'Which exercise is best?' is not appropriate. These exercise modalities are not competitors but collaborators. A truly effective slow-aging protocol must integrate all three to build a multi-faceted defense system of telomere maintenance (aerobic), mitochondrial health (HIIT), and structural integrity and metabolic function (resistance exercise). This is a practical strategy derived from a high-level synthesis of the provided data.

Table 2: Comparative Effects of Exercise Types on Cellular Aging Indicators
Exercise Type
Aerobic Exercise (AE)
Resistance Exercise (RE)
High-Intensity Interval Training (HIIT)

4.3. Pillar 3: The Mind-Body Axis, Stress, Sleep, and the Pace of Aging

4.3.1. The Biology of Stress-Induced Aging

Mental stress is not just a psychological issue; it is a powerful biological factor that directly accelerates cellular aging. Chronic stress continuously activates the hypothalamic-pituitary-adrenal (HPA) axis, leading to chronically elevated levels of the stress hormone **cortisol**.[1]

Excessive cortisol breaks down muscle and stores energy as visceral fat, disrupting the metabolic system.[1] Furthermore, chronically high cortisol levels induce a state of systemic

low-grade chronic inflammation and **oxidative stress**, which act as key mechanisms that directly damage DNA and accelerate **telomere shortening**.[53] Ultimately, chronic stress weakens immune function and acts as a trigger that exacerbates almost all hallmarks of aging.

4.3.2. Sleep: The Essential Condition for Recovery

Sleep is not merely a time for rest but an essential recovery process that repairs cells damaged during the day and resets bodily systems.[56] If the quality or quantity of sleep is poor, this recovery process is insufficient, and aging is accelerated. Studies show that sleep deprivation is directly associated with shorter telomeres, increased inflammation levels, and impaired cellular repair processes.[57]

Insufficient sleep disrupts the immune and metabolic processes, creating an environment that promotes cellular aging.[56] As the DNA repair, growth hormone secretion, and inflammation regulation processes that should occur overnight are disturbed, our bodies are set on a path of accelerated aging.

4.3.3. Evidence-Based Mitigation Strategies

- **Stress Management:** Techniques such as **Mindfulness-Based Stress Reduction (MBSR), meditation, and deep breathing** have been scientifically proven to suppress HPA axis overactivation, lower cortisol levels, and reduce inflammation.[61] Additionally, regular exercise and close social relationships serve as the most powerful buffers against the negative effects of stress.[55]
- **Sleep Hygiene:** Adhering to evidence-based sleep hygiene practices—such as going to bed and waking up at the same time every day, keeping the bedroom dark and cool, and avoiding electronic devices before sleep—is crucial for maximizing cellular recovery time and improving sleep quality.

V. Identifying and Mitigating Aging Accelerators

5.1. Systemic Accelerators: Inflammation and Metabolic Dysfunction

Modern lifestyles include several accelerators that directly trigger the hallmarks of aging.

- **Chronic Hyperglycemia and Insulin Resistance:** The excessive consumption of refined carbohydrates is a primary cause of chronic hyperglycemia and insulin resistance, which leads to 'deregulated nutrient-sensing.' High blood sugar non-enzymatically binds to proteins to form **Advanced Glycation End-products (AGEs)**, which cross-link proteins like collagen, making tissues stiff and dysfunctional.[12] A loss of skin elasticity (so-called 'sugar face') or hardening of the arteries are prime examples.
- **Smoking:** Smoking is one of the most potent aging accelerators, injecting a massive amount of oxidants into the body, causing systemic inflammation, vascular damage, and DNA mutations.[64] Smoking directly exacerbates virtually all hallmarks of aging.
- **Excessive Alcohol Consumption:** The process of alcohol metabolism produces acetaldehyde, a toxic substance, and generates a large amount of reactive oxygen species, causing oxidative stress. Excessive drinking depletes antioxidants and has been directly confirmed to accelerate telomere shortening.[70]

5.2. Environmental Accelerators: A Focused Analysis of Photoaging

Ultraviolet (UV) radiation is the most powerful external environmental factor that accelerates skin aging.[70] The key molecular mechanisms of photoaging are as follows:

1. Upon UV exposure, a large amount of **reactive oxygen species (ROS)** is generated in skin cells (oxidative stress).
2. The generated ROS activates signaling pathways that increase the expression of **Matrix Metalloproteinases (MMPs)**.
3. MMPs **degrade collagen and elastin**, the structural supports of the skin, causing wrinkles and loss of elasticity.
4. Simultaneously, UV radiation **directly damages cellular DNA**, causing mutations and increasing the risk of skin cancer.

Topical intervention methods to counter this photoaging include:

- **Sunscreen:** The most basic first line of defense against photoaging.
- **Topical Retinoids:** Vitamin A derivatives that normalize cell turnover, promote new collagen synthesis, and inhibit the activity of MMPs, thereby directly improving the signs of photoaging.[78]
- **Moisturizers (Ceramides & Hyaluronic Acid):** Ceramides and hyaluronic acid strengthen the skin barrier function, which is weakened by intrinsic aging and UV damage. A strong skin barrier is essential for preventing moisture loss and protecting the

skin from harmful external environments.[84]

VI. Synthesis and Recommendations: An Integrated Framework for Slow Aging

6.1. The Integrated Slow-Aging Protocol

Synthesizing all the information presented in this report, it is clear that slow aging can be achieved not through a sum of fragmented efforts but through an integrated strategy that spans one's entire lifestyle. Each pillar is important individually, but they are organically connected and create a synergistic effect. For example, regular exercise improves insulin sensitivity, maximizing the effects of diet, while also serving as a powerful stress reliever.

The claim that slow aging is possible with just one specific food or supplement lacks scientific evidence.[1] Slow aging is an emergent property of a complex system created by the harmonious interaction of the four pillars: diet, exercise, sleep, and stress management.

6.2. Hormesis as a Unifying Principle

Underlying many slow-aging strategies is a unified scientific principle called **Hormesis**. Hormesis is the phenomenon where an organism, when exposed to a moderate level of stress, activates its defense and repair systems, becoming healthier and stronger in the long run.[40]

- **Exercise:** Imposes acute physical and metabolic stress to induce long-term adaptation and recovery.
- **Fasting:** Activates cellular cleanup and repair pathways through temporary energy deficit stress.
- **Phytonutrients:** Mildly toxic substances (like polyphenols) that plants produce to protect themselves act as beneficial stressors in the human body, stimulating its defense systems.

In essence, the key to slow aging is not to avoid all stress but to periodically apply beneficial stress at a recoverable level to train our body's intrinsic recovery and defense systems.

6.3. Concluding Perspective: The Future of Proactive, Preventive Medicine

In conclusion, slow aging is no longer a niche interest but is establishing itself as a core paradigm of proactive and preventive future medicine. By understanding the fundamental biological hallmarks of aging and targeting them, we can move away from a passive approach of treating age-related diseases after they occur to a proactive health management that prevents the onset of diseases and maintains a high level of function throughout life. The ultimate goal of slow aging is for everyone to align their healthspan with their lifespan, realizing a life that is both 'long and well-lived'.[1]

참고 자료

1. “느리게 나이 들고 싶다면 '노잼' 활동 즐기세요” | 신동아, 8월 21, 2025에 액세스, https://shindonga.donga.com/culture/article/all/13/5519974/1
2. 느리게 나이 드는 습관 + 당신도 느리게 나이 들 수 있습니다 - 예스24, 8월 21, 2025에 액세스, https://m.yes24.com/Goods/Detail/124408728
3. The hallmarks of aging as a conceptual framework for health and longevity research, 8월 21, 2025에 액세스, https://www.frontiersin.org/journals/aging/articles/10.3389/fragi.2024.1334261/full
4. 피할 수 없다면 맞서라, 노화의 종말 - KPMG International, 8월 21, 2025에 액세스, https://assets.kpmg.com/content/dam/kpmg/kr/pdf/2024/business-focus/kpmg-korea-anti-aging_20240510.pdf
5. 노인학 전문의가 말하는 '노화지연'... 젊음 유지하는 4가지 건강법 소개 - 동아일보, 8월 21, 2025에 액세스, https://www.donga.com/news/It/article/all/20230117/117475225/1
6. 지속가능한 나이듦(리커버) | 정희원 - 교보문고, 8월 21, 2025에 액세스, https://product.kyobobook.co.kr/detail/S000201274253
7. The Hallmarks of Aging - PMC, 8월 21, 2025에 액세스, https://pmc.ncbi.nlm.nih.gov/articles/PMC3836174/
8. Hallmarks of aging - Wikipedia, 8월 21, 2025에 액세스, https://en.wikipedia.org/wiki/Hallmarks_of_aging
9. Hallmarks of Aging - Sigma-Aldrich, 8월 21, 2025에 액세스, https://www.sigmaaldrich.com/deepweb/assets/sigmaaldrich/product/documents/348/609/pb1431en-ms.pdf
10. Targeting the hallmarks of aging: mechanisms and therapeutic opportunities - PMC, 8월 21, 2025에 액세스, https://pmc.ncbi.nlm.nih.gov/articles/PMC12259695/
11. Hallmarks of Skin Aging: Update, 8월 21, 2025에 액세스, https://www.aginganddisease.org/EN/10.14336/AD.2023.0321

12. Aging Hallmarks and Progression and Age-Related Diseases: A Landscape View of Research Advancement - ACS Publications, 8월 21, 2025에 액세스, https://pubs.acs.org/doi/10.1021/acschemneuro.3c00531
13. Hallmarks of aging: An expanding universe - PubMed, 8월 21, 2025에 액세스, https://pubmed.ncbi.nlm.nih.gov/36599349/
14. 12 Hallmarks of Aging: What They Mean for Your Health - Jinfiniti, 8월 21, 2025에 액세스, https://www.jinfiniti.com/hallmarks-of-aging/
15. Decoding the 12 Hallmarks of Aging - OneSkin, 8월 21, 2025에 액세스, https://www.oneskin.co/blogs/reference-lab/hallmarks-of-aging
16. 건강정보 2 페이지 - 닥터박민수닷컴, 8월 21, 2025에 액세스, https://drparkminsu.com/health_information?page=2
17. In Vivo Amelioration of Age-Associated Hallmarks by Partial Reprogramming - PMC, 8월 21, 2025에 액세스, https://pmc.ncbi.nlm.nih.gov/articles/PMC5679279/
18. Old human cells rejuvenated with stem cell technology - Stanford Medicine, 8월 21, 2025에 액세스, https://med.stanford.edu/news/all-news/2020/03/old-human-cells-rejuvenated-with-stem-cell-technology.html
19. Cellular rejuvenation therapy safely reverses signs of aging in mice ..., 8월 21, 2025에 액세스, https://www.salk.edu/news-release/cellular-rejuvenation-therapy-safely-reverses-signs-of-aging-in-mice/
20. Scientists have shown ageing process can be safely and effectively reversed in mice, 8월 21, 2025에 액세스, https://www.drugtargetreview.com/news/101826/scientists-have-shown-ageing-process-can-be-safely-and-effectively-reversed-in-mice/
21. Cellular Rejuvenation Therapy Reverses Mouse Aging in a Safe Way - NAD.com, 8월 21, 2025에 액세스, https://www.nad.com/news/cellular-rejuvenation-therapy-safely-reverses-mouse-aging
22. Healthy Aging - Salk Institute, 8월 21, 2025에 액세스, https://www.salk.edu/science/research/aging-and-regenerative-medicine/news/
23. 150 년까지 생활하는 것이 가능할 수 있습니다. 최신 연구에서 답을 찾았을 것입니다. - Infobae, 8월 21, 2025에 액세스, https://www.infobae.com/kr/2022/03/25/living-to-150-years-may-be-possible-the-latest-research-would-have-found-the-answer/
24. Top 10 longevity studies of 2024 - Elysium Health, 8월 21, 2025에 액세스, https://www.elysiumhealth.com/blogs/aging101/top-10-longevity-studies-of-2024
25. Dietary Polyphenols as Anti-Aging Agents: Targeting the Hallmarks of Aging - MDPI, 8월 21, 2025에 액세스, https://www.mdpi.com/2072-6643/16/19/3305
26. Science is making anti-aging progress. But do we want to live forever? - Harvard Gazette, 8월 21, 2025에 액세스, https://news.harvard.edu/gazette/story/2024/05/science-is-making-anti-aging-progress-but-do-we-want-to-live-forever/
27. “노화도 질병, 치료해 110세까지”...수퍼센티네리언 시대 온다 - 조선일보, 8월 21,

2025에 액세스,
https://www.chosun.com/economy/weeklybiz/2024/08/15/JO75RMO4FRARPHUPUKMRQQ6HLI/
28. Life expectancy may be reaching upper limits—for now, 8월 21, 2025에 액세스, https://hsph.harvard.edu/news/life-expectancy-may-be-reaching-upper-limits-for-now/
29. The ups and downs of caloric restriction and fasting: from molecular effects to clinical application - PubMed Central, 8월 21, 2025에 액세스, https://pmc.ncbi.nlm.nih.gov/articles/PMC8749464/
30. Caloric restriction and intermittent fasting: Two potential diets for successful brain aging - PMC - PubMed Central, 8월 21, 2025에 액세스, https://pmc.ncbi.nlm.nih.gov/articles/PMC2622429/
31. Calorie Restriction and Aging in Humans - PMC, 8월 21, 2025에 액세스, https://pmc.ncbi.nlm.nih.gov/articles/PMC9042193/
32. Intermittent and periodic fasting, longevity and disease - PMC - PubMed Central, 8월 21, 2025에 액세스, https://pmc.ncbi.nlm.nih.gov/articles/PMC8932957/
33. The Review of Anti-aging Mechanism of Polyphenols on Caenorhabditis elegans - Frontiers, 8월 21, 2025에 액세스, https://www.frontiersin.org/journals/bioengineering-and-biotechnology/articles/10.3389/fbioe.2021.635768/full
34. The Review of Anti-aging Mechanism of Polyphenols on Caenorhabditis elegans, 8월 21, 2025에 액세스, https://www.researchgate.net/publication/352905164_The_Review_of_Anti-aging_Mechanism_of_Polyphenols_on_Caenorhabditis_elegans
35. The Molecular Mechanism of Polyphenols in the Regulation of ..., 8월 21, 2025에 액세스, https://pmc.ncbi.nlm.nih.gov/articles/PMC10049696/
36. Dietary Anti-Aging Polyphenols and Potential Mechanisms - PMC - PubMed Central, 8월 21, 2025에 액세스, https://pmc.ncbi.nlm.nih.gov/articles/PMC7918214/
37. Plant polyphenols: The secret to living longer and healthy aging? - News-Medical, 8월 21, 2025에 액세스, https://www.news-medical.net/news/20241002/Plant-polyphenols-The-secret-to-living-longer-and-healthy-aging.aspx
38. Can the Blue Zone Diet Help You Live Longer? - American Institute ..., 8월 21, 2025에 액세스, https://www.aicr.org/resources/blog/can-the-blue-zone-diet-help-you-live-longer/
39. Effects of exercise on cellular and tissue aging - PMC - PubMed Central, 8월 21, 2025에 액세스, https://pmc.ncbi.nlm.nih.gov/articles/PMC8202894/
40. Energetic Interventions for healthspan and resiliency with aging - PMC - PubMed Central, 8월 21, 2025에 액세스, https://pmc.ncbi.nlm.nih.gov/articles/PMC5133182/
41. Molecular mechanisms of life- and health-span extension: role of calorie restriction and exercise intervention - PubMed, 8월 21, 2025에 액세스, https://pubmed.ncbi.nlm.nih.gov/18059622/
42. Exercise delays aging: evidence from telomeres and telomerase —a systematic review and meta-analysis of randomized controlled trials - Frontiers, 8월 21,

2025에 액세스, https://www.frontiersin.org/journals/physiology/articles/10.3389/fphys.2025.1627292/full
43. Systematic Review and Meta-analysis Highlights a Link Between Aerobic Fitness and Telomere Maintenance | The Journals of Gerontology - Oxford Academic, 8월 21, 2025에 액세스, https://academic.oup.com/biomedgerontology/article/80/6/glaf068/8115838
44. Effects of Physical Exercise ... - JMIR Public Health and Surveillance, 8월 21, 2025에 액세스, https://publichealth.jmir.org/2024/1/e46019
45. "The Effect of High-Intensity Interval Training (HIIT) on Epigenetic Aging Markers", 8월 21, 2025에 액세스, https://openpublichealthjournal.com/VOLUME/18/ELOCATOR/e18749445363042/
46. "The Effect of High-Intensity Interval Training (HIIT) on Epigenetic Aging Markers" - The Open Public Health Journal, 8월 21, 2025에 액세스, https://openpublichealthjournal.com/VOLUME/18/ELOCATOR/e18749445363042/PDF/
47. High Intensity Training and Aging: Mayo Clinic Radio - YouTube, 8월 21, 2025에 액세스, https://www.youtube.com/watch?v=0p9Bz65UIFs
48. Mayo Clinic discovers high-intensity aerobic training can reverse ..., 8월 21, 2025에 액세스, https://newsnetwork.mayoclinic.org/discussion/mayo-clinic-discovers-high-intensity-aerobic-training-can-reverse-aging-processes-in-adults/
49. Healing with HIIT Exercises: Cellular Renewal & Anti-inflammatory Responses, 8월 21, 2025에 액세스, https://amitray.com/healing-with-hiit-exercises-longevity-protocols/
50. Stress Hormones: Unveiling the Role in Accelerated Cellular Senescence, 8월 21, 2025에 액세스, https://www.aginganddisease.org/EN/10.14336/AD.2024.0262
51. Psychological Stress and Cellular Aging - Oxford Research Encyclopedias, 8월 21, 2025에 액세스, https://oxfordre.com/psychology/oso/viewentry/10.1093$002facrefore$002f9780190236557.001.0001$002facrefore-9780190236557-e-131
52. Adrenal Aging and Its Implications on Stress Responsiveness in Humans - Frontiers, 8월 21, 2025에 액세스, https://www.frontiersin.org/journals/endocrinology/articles/10.3389/fendo.2019.00054/full
53. The Link between Chronic Stress and Accelerated Aging - PMC, 8월 21, 2025에 액세스, https://pmc.ncbi.nlm.nih.gov/articles/PMC7400286/
54. Evidence of Youthful Aging: Chronic Stress and the Association With DNA Damage - UNI ScholarWorks, 8월 21, 2025에 액세스, https://scholarworks.uni.edu/cgi/viewcontent.cgi?article=1000&context=mcnairsymposium
55. How chronic stress is harming our DNA - American Psychological Association, 8월 21, 2025에 액세스, https://www.apa.org/monitor/2014/10/chronic-stress
56. Sleep and Biological Aging: A Short Review - PMC, 8월 21, 2025에 액세스, https://pmc.ncbi.nlm.nih.gov/articles/PMC8658028/

57. The association between sleep quality and telomere length: A systematic literature review, 8월 21, 2025에 액세스, https://research.bond.edu.au/files/215645969/The_association_between_sleep_quality_and_telomere_length_A_systematic_literature_review.pdf
58. The association between sleep quality and telomere length: A systematic literature review, 8월 21, 2025에 액세스, https://www.researchgate.net/publication/366981032_The_association_between_sleep_quality_and_telomere_length_A_systematic_literature_review
59. How Sleep Affects Your Telomeres | Psychology Today, 8월 21, 2025에 액세스, https://www.psychologytoday.com/us/blog/sleep-newzzz/202107/how-sleep-affects-your-telomeres
60. The association between sleep quality and telomere length: A systematic literature review - PMC - PubMed Central, 8월 21, 2025에 액세스, https://pmc.ncbi.nlm.nih.gov/articles/PMC9860369/
61. 11 Stress Management Tips for Older Adults — Talkspace, 8월 21, 2025에 액세스, https://www.talkspace.com/blog/stress-management-for-older-adults/
62. Stress Management Tips for Older Adults - Miami Jewish Health, 8월 21, 2025에 액세스, https://www.miamijewishhealth.org/blog/senior-health-wellness/stress-management-tips-for-older-adults/
63. The Impact of Stress on Older Adults and Tips for Managing It | Hebrew SeniorLife, 8월 21, 2025에 액세스, https://www.hebrewseniorlife.org/blog/impact-stress-older-adults-and-tips-managing-it
64. Oxidative stress and accelerated vascular aging: implications for ..., 8월 21, 2025에 액세스, https://pmc.ncbi.nlm.nih.gov/articles/PMC2756477/
65. Oxidative stress and accelerated vascular aging: implications for cigarette smoking, 8월 21, 2025에 액세스, https://www.imrpress.com/journal/fbl/14/8/10.2741/3440
66. Cigarette smoking drives accelerated aging across human tissues - bioRxiv, 8월 21, 2025에 액세스, https://www.biorxiv.org/content/10.1101/2024.03.14.585016v1.full-text
67. Cigarette Smoke-Induced Reactive Oxygen Species Formation: A Concise Review - MDPI, 8월 21, 2025에 액세스, https://www.mdpi.com/2076-3921/12/9/1732
68. Relationships among smoking, oxidative stress, inflammation, macromolecular damage, and cancer - eScholarship, 8월 21, 2025에 액세스, https://escholarship.org/content/qt92g4d9hp/qt92g4d9hp_noSplash_217d9de76836331880dbd4a621527733.pdf
69. The Impact of Tobacco Cigarettes, Vaping Products and Tobacco Heating Products on Oxidative Stress - MDPI, 8월 21, 2025에 액세스, https://www.mdpi.com/2076-3921/11/9/1829
70. Shortened telomeres in individuals with abuse in alcohol ..., 8월 21, 2025에 액세스, https://pmc.ncbi.nlm.nih.gov/articles/PMC3125427/
71. Alcohol has a dramatic impact on your biological age. Here's how, 8월 21, 2025에 액세스, https://www.sciencefocus.com/the-human-body/alcohol-biological-age

72. Mechanism of skin photoaging caused by UVA and UVB. UVA (315–400 nm) - ResearchGate, 8월 21, 2025에 액세스, https://www.researchgate.net/figure/Mechanism-of-skin-photoaging-caused-by-UVA-and-UVB-UVA-315-400-nm-UVA-penetrates-the_fig1_357784713
73. Antioxidants in Photoaging: From Molecular Insights to Clinical Applications - MDPI, 8월 21, 2025에 액세스, https://www.mdpi.com/1422-0067/25/4/2403
74. A Comprehensive Review of the Role of UV Radiation in ..., 8월 21, 2025에 액세스, https://pmc.ncbi.nlm.nih.gov/articles/PMC12018068/
75. Mechanisms of Photoaging and Chronological Skin Aging | Request PDF - ResearchGate, 8월 21, 2025에 액세스, https://www.researchgate.net/publication/11031343_Mechanisms_of_Photoaging_and_Chronological_Skin_Aging
76. Ultraviolet Radiation, Aging and the Skin: Prevention of Damage by Topical cAMP Manipulation - MDPI, 8월 21, 2025에 액세스, https://www.mdpi.com/1420-3049/19/5/6202
77. Photoaging (Sun Damage) > Fact Sheets > Yale Medicine, 8월 21, 2025에 액세스, https://www.yalemedicine.org/conditions/sun-damage
78. What is the recommended approach for using topical retinoids (retinoic acid) for anti-aging?, 8월 21, 2025에 액세스, https://www.droracle.ai/articles/157788/topical-retinoids-for-anti-aging
79. Human Skin Aging and the Anti-Aging Properties of Retinol - MDPI, 8월 21, 2025에 액세스, https://www.mdpi.com/2218-273X/13/11/1614
80. Retinoids in the treatment of skin aging: an overview of clinical ..., 8월 21, 2025에 액세스, https://pmc.ncbi.nlm.nih.gov/articles/PMC2699641/
81. The Chemistry of Beauty: How Retinoids Enhance Skin Health - News-Medical, 8월 21, 2025에 액세스, https://www.news-medical.net/health/The-Chemistry-of-Beauty-How-Retinoids-Enhance-Skin-Health.aspx
82. Tretinoin: Uses, Interactions, Mechanism of Action | DrugBank Online, 8월 21, 2025에 액세스, https://go.drugbank.com/drugs/DB00755
83. Topical retinoids (vitamin a creams) - DermNet, 8월 21, 2025에 액세스, https://dermnetnz.org/topics/topical-retinoids
84. Ceramides & Hyaluronic Acid: Their Role in Achieving ... - CeraVe, 8월 21, 2025에 액세스, https://www.cerave.com.au/blog/everyday-skin-care/ceramides-and-hyaluronic-acid
85. Can You Use Ceramides and Hyaluronic Acid Together? - Kinship, 8월 21, 2025에 액세스, https://lovekinship.com/blogs/news/ceramides-and-hyaluronic-acid
86. What's Better? Ceramides or Hyaluronic Acid? - Aesthetics by Design, 8월 21, 2025에 액세스, https://aestheticsbydesign.com/news-blog/2022/2/25/whats-better-ceramides-or-hyaluronic-acid
87. The Best Moisturizers for Aging Skin: The 7 Ingredients That Matter - Midi Health, 8월 21, 2025에 액세스, https://www.joinmidi.com/post/best-moisturizer-for-aging-skin

88. Skin Hydration: The Roles of Hyaluronic Acid and Ceramides - Vanicream, 8월 21, 2025에 액세스, https://www.vanicream.com/skin-basics/skin-hydration
89. Ceramides and Aging: Can They Help Reverse the Signs of Aging - Blue Nectar, 8월 21, 2025에 액세스, https://www.bluenectarproduct.com/blogs/beauty-wellness-articles/ceramides-and-aging-can-they-help-reverse-the-signs-of-aging