

***A Review on Emerging Anti-Aging Strategies Scientific Basis
and Efficacy***



Daffodil
International
University

PROJECT REPORT

[In the partial fulfillment of the requirements for the degree of Bachelor of
Pharmacy]

Submitted To

The Department of Pharmacy
Faculty of Health & Life Sciences
Daffodil International University

Submitted By

Fatema Tuz Zohura Elma
Student ID: 201-29-1650
Department of Pharmacy
Faculty of Health & Life Sciences
Daffodil International University

.....

April 2024

APPROVAL

This project paper, “**A Review on Emerging Anti-Aging Strategies Scientific Basis and Efficacy**”, submitted to the Department of Pharmacy, Faculty of Health & Life Sciences, Daffodil International University, has been accepted as satisfactory for the partial fulfillment of the requirements for the degree of Bachelor of Pharmacy and approved as to its style and contents.

BOARD OF EXAMINERS

.....

Dr. Muniruddin Ahmed
Professor and Head
Department of Pharmacy
Faculty of Health & Life Sciences
Daffodil International University

.....

Internal Examiner 1

.....

Internal Examiner 2

.....

External Examiner 3

DECLARATION

I hereby declare that this project report, “**A Review on Emerging Anti-Aging Strategies Scientific Basis and Efficacy**” is done by me under the supervision of Dr. Sharifa Sultana Associate Professor and Associate Head, I am declaring that this Project is my original work. I also declare that neither this project nor any part thereof has been submitted elsewhere for the award of Bachelor or any degree.

Supervised by



.....

Dr. Sharifa Sultana

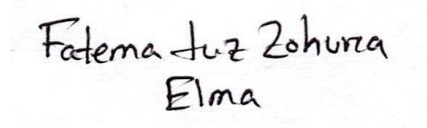
Associate Professor and Associate Head

Department of Pharmacy

Faculty of Health & Life Sciences

Daffodil International University

Submitted By



.....

Fatema Tuz Zohura Elma

Student ID: 201-29-1650

Department of Pharmacy

Faculty of Allied Health Sciences

Daffodil International University

ACKNOWLEDGEMENT

I might want to communicate my profound applause to the All-powerful Allah who has given me the capacity to finish my undertaking work and the chance to concentrate in this subject.

I'm a lot of thankful to my honorable project supervisor Dr. Sharifa Sultana Associate Professor and Associate Head, Department of Pharmacy, Daffodil International University.

I would like to express my humble regards to Dr. Muniruddin Ahmed, Professor and Head, Department of Pharmacy, Daffodil International University.

I also wish to offer my respect to all of the teachers of Pharmacy Department, Daffodil International University and thankful to other members for their excellent cooperation with us.

Finally, I would like to express my gratitude towards my parents and other family members for their kind cooperation and encouragement which helped me in completion of this project.



My Parents,

The persons who always encourage me in every sphere of my life.

My teacher,

The persons who guided me in this process and the committee who kept me on track.

Abstract

Although people are living longer, there is an increasing tendency in the incidence of age-related diseases. At the moment, one of the main causes of morbidity and death worldwide is age-related illnesses. Finding appropriate therapies that can slow back aging and lower or delay the occurrence of crippling illnesses associated with aging is therefore desperately needed. The effectiveness of new anti-aging techniques for preserving better health as one ages is covered in this assessment. Numerous anti-aging techniques are being developed; these include actions like increasing autophagy, removing senescent cells, transferring plasma from young blood, fasting occasionally, increasing adult neurogenesis, engaging in physical activity, consuming antioxidants, and using stem cell treatment. Several preliminary investigations indicate that taking doses of BDNF, senolytic drugs, young blood plasma, autophagy enhancement factors, and medications that boost neurogenesis represent exciting strategies to maintain normal health as we age and delay age-related neurodegenerative diseases like Alzheimer's disease. The ability of stem cell therapy to promote brain regrowth and functioning in elderly or Alzheimer's disease patients has also showed promise. A number of these strategies are awaiting a thorough evaluation in clinical trials to ascertain their long-term effectiveness and any drawbacks. However, non-invasive methods like intermittent fasting, exercise, and the consumption of antioxidants like curcumin and resveratrol have demonstrated great promise in enhancing function during the aging process. Some of these methods are even ready for large-scale clinical studies. In conclusion, a number of strategies are poised to emerge as commonplace treatments in the years to come for slowing down the aging process and delaying age-related illnesses.

Contents

Chapter 1.....	1
Introduction	1
1. Introduction	2
1.1 Etiology of Aging	3
1.2 Ecology and the aging process	5
1.3 Preventive action of Aging	7
Chapter 2.....	9
Literature Review	9
2.1 Skin anti-aging strategies.....	10
2.2 Skin Aging & Modern Age Anti-Aging Strategies	10
2.3 Current Trends in Anti-Aging Strategies.....	11
Chapter 3.....	12
Purpose of the study	12
3.1 Purpose of the study	13
Chapter 4.....	14
Methodology.....	14
4.1 Data collection procedure	15
4.2 Data analysis strategy	15
Chapter 5.....	16
Results & Discussion.....	16
5.1 Autophagy enhancement for facilitating successful aging	17
5.2 Senescent cell elimination as an anti-aging therapy	18
5.3 Intermittent fasting as a means to combat aging	20
5.3.1 Satisfaction level of the consumer with antiaging product used	21

5.3.2 Duration of use of the participants of antiaging cosmetics	22
5.4 Photo protection and Systemic Antioxidants.....	22
5.5 Topical Pharmacological Agents with Anti-Aging Properties	24
5.6 Stem cell therapy for promoting healthy brain aging and reversing AD.....	24
Chapter 6.....	26
Conclusion	26
6.1 Conclusion	27
Chapter 7.....	28
Reference	28

List of figures

Figure 1: Pathogenesis of aging.....	5
Figure 2: Influence of Normal Aging on Brain Autophagy.....	18
Figure 3: Overview of various antiaging strategies	19
Figure 4: Satisfaction level of the consumer with antiaging product used	21
Figure 5: Duration of use of the participants of antiaging cosmetics	22

List of tables

Table 1: Skin antiaging approach	21
Table 2: Available antioxidant for anti-aging with mechanism.....	24

Chapter 1

Introduction

1. Introduction

The field of anti-aging investigation has seen an incredible development in the quest to preserve youthful vigor and mitigate the ravages of aging. Growing interest has been observed for comprehending the biological processes behind aging and creating techniques to counteract their consequences, thanks to scientific and technological developments. Many different types of people have become interested in the search for anti-aging therapies, which range from skincare products to lifestyle changes [1]. "Emerging Anti-Aging Strategies: Scientific Basis and Efficacy" examines the most recent advancements in the field of anti-aging therapies, examining the theoretical underpinnings and assessing the effectiveness of cutting-edge methods. Investigators are finding complex biological mechanisms linked to aging in this age of unparalleled scientific discovery, opening the door for novel therapeutic approaches that address the underlying causes of age-related deterioration [2]. In the last century, human life duration has nearly doubled in affluent nations. Nonetheless, populations' increased longevity has led to a higher prevalence of age-related illnesses. Globally, age-related illnesses like cancer, neurological diseases, and cardiovascular disease are the main causes of morbidity and mortality. Over the course of an organism's lifetime, aging of cells, tissues, and organs along with a continuous decline in effectiveness are normal occurrences [3]. This comprehensive review provides insights into the multifaceted nature of aging, encompassing genetic predispositions, environmental factors, and lifestyle choices. By elucidating the interplay between various determinants of aging, researchers can identify promising avenues for intervention, ranging from pharmaceutical interventions to dietary supplements and beyond. Through a critical examination of current research findings, this review aims to shed light on the potential benefits and limitations of emerging anti-aging strategies [4]. By synthesizing evidence from preclinical studies, clinical trials, and epidemiological research, we aim to provide a balanced perspective on the efficacy and safety of anti-aging interventions, empowering both consumers and healthcare professionals to make informed decisions. Moreover, this review underscores the importance of adopting a holistic approach to anti-aging, recognizing the interconnectedness of biological, psychological, and social factors [5]. Beyond targeting individual biomarkers of aging, effective anti-aging strategies should encompass lifestyle modifications, stress management techniques, and social support

systems to promote overall well-being and resilience against age-related decline. As we embark on this journey through the frontiers of anti-aging research, it is essential to maintain a critical mindset, acknowledging both the potential promises and pitfalls of emerging interventions. By fostering collaboration between researchers, clinicians, and policymakers, we can accelerate the translation of scientific discoveries into tangible benefits for aging populations worldwide [6]. Certain anti-aging techniques that increase longevity might also be helpful in postponing the appearance of age-related illnesses. The effectiveness of new anti-aging techniques for preserving better health in old age is demonstrated by this overview. We'll talk about approaches that have attracted a lot of attention recently. These involve techniques like boosting autophagy, utilizing senolytics to eliminate senescent cells, transfusion of young blood, intermittent fasting, and increasing adult neurogenesis, engaging in physical activity, consuming antioxidants and herbal supplements, and receiving stem cell therapy [7].

1.1 Etiology of Aging

Biology and medicine have devoted a great deal of research to the intricate and multidimensional subject of the epidemiology of aging, or the causes of aging. Even while aging is a normal process that happens to all living things, its basic processes are still unclear [7]. On the other hand, a number of theories have been put forth to explain the genesis of aging:

1. **Genetic Theories:** These theories propose that aging is programmed into our genes and is therefore predetermined. One example is the "Telomere Shortening Theory," which suggests that the shortening of telomeres (the protective caps at the end of chromosomes) with each cell division contributes to cellular aging and eventual death.
2. **Cellular Damage Theories:** These theories suggest that aging is a result of accumulated damage to cells and tissues over time. This damage can be caused by various factors, including oxidative stress, DNA damage, protein misfolding, and inflammation. The "Free Radical Theory of Aging" proposes that accumulated damage from reactive oxygen species (ROS) leads to cellular dysfunction and aging [8].
3. **Hormonal Theories:** Hormonal changes over time are thought to play a significant role in the aging process. For instance, declines in hormone levels such as growth hormone,

estrogen, and testosterone are associated with aging-related changes in metabolism, muscle mass, and other physiological functions.

4. **Mitochondrial Dysfunction:** Mitochondria are the powerhouses of the cell, responsible for producing energy. The "Mitochondrial Theory of Aging" suggests that age-related damage to mitochondria and decline in their function contribute to cellular aging and overall organismal decline [9].
5. **Immune System Decline:** Aging is associated with a decline in immune function, a phenomenon known as immunosenescence. This decline makes older individuals more susceptible to infections, autoimmune diseases, and cancer [10].
6. **Epigenetic Changes:** Epigenetic modifications, such as DNA methylation and histone modifications, regulate gene expression without changing the underlying DNA sequence. Changes in epigenetic regulation have been implicated in aging and age-related diseases.
7. **Caloric Restriction and Longevity:** Studies have shown that reducing calorie intake without malnutrition can extend lifespan in various organisms, including mammals. This suggests that metabolic factors play a role in the aging process.
8. **Altered Intercellular Communication:** Communication between cells plays a crucial role in maintaining tissue homeostasis. Age-related changes in intercellular communication, such as increased inflammation and altered signaling pathways, contribute to tissue dysfunction and aging [11].

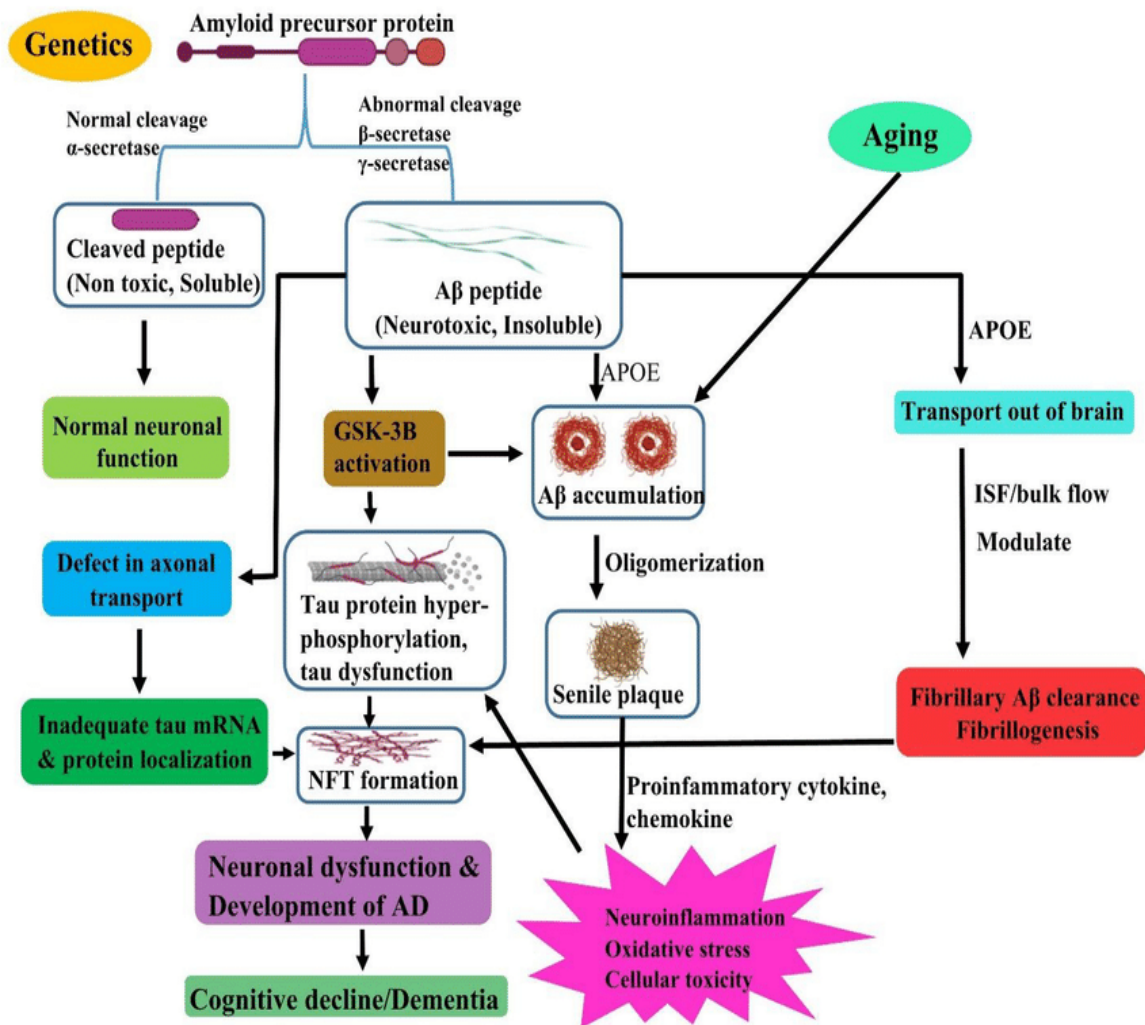


Figure 1: Pathogenesis of aging

1.2 Ecology and the aging process

Ecology, the investigation of how organisms communicate with their surroundings, offers important insights into how populations and individuals age. The intricate process of aging, which encompasses genetic, physiological, and environmental elements, is impacted by ecological dynamics at multiple scales, ranging from individual species to entire ecosystems. Comprehending these ecological factors can illuminate the workings of aging and provide guidance for tactics aimed at encouraging healthy aging in humans and other living things [12].

Population Dynamics: In ecological studies, populations of organisms are often analyzed in terms of birth rates, death rates, and migration patterns. Similarly, aging populations are

characterized by changes in birth rates (fertility), death rates (mortality), and migration (movement of individuals in and out of the population). Ecological concepts such as carrying capacity, which refers to the maximum population size that an environment can sustainably support, can be applied to understand the dynamics of aging populations within their environments [13].

Resource Availability: Ecological principles emphasize the importance of resource availability in determining population growth and survival. In the context of aging, access to resources such as food, water, shelter, and healthcare can significantly influence the health and longevity of individuals. Competition for limited resources may exacerbate the effects of aging, particularly in environments where resources are scarce or unevenly distributed.

Habitat Quality: The quality of the habitat in which organisms live plays a crucial role in their health and survival. Ecological studies have shown that exposure to pollutants, habitat degradation, and loss of biodiversity can negatively impact the health of individuals and populations. Similarly, environmental factors such as air and water quality, climate change, and habitat fragmentation can affect the aging process by increasing susceptibility to age-related diseases and reducing overall life expectancy [14].

Interactions with Other Species: Ecological communities consist of diverse species interacting with each other in complex ways. Similarly, the aging process can be influenced by interactions with other species, including pathogens, parasites, predators, and mutualistic organisms. For example, exposure to infectious diseases can accelerate aging by compromising immune function, while symbiotic relationships with beneficial microbes may promote healthy aging by supporting physiological functions such as digestion and immune regulation.

Adaptation and Resilience: Ecological systems exhibit resilience, the ability to withstand and recover from disturbances. Similarly, individuals vary in their capacity to adapt to the challenges of aging, influenced by genetic predispositions, lifestyle choices, and environmental factors. Understanding the factors that contribute to resilience in both ecological and aging contexts can inform interventions aimed at enhancing adaptive capacity and promoting successful aging [15].

Conservation and Sustainability: Ecological principles emphasize the importance of conservation and sustainability to maintain healthy ecosystems and biodiversity. Similarly, efforts to promote healthy aging involve strategies aimed at preserving the health and well-being of aging populations. By integrating ecological principles into public health policies and practices, societies can work towards creating environments that support healthy aging for individuals and communities [16].

1.3 Preventive action of Aging

The topic of preventing aging is intricate and multidimensional, involving genetics, medical treatments, and different lifestyle choices. Although there is no way to completely stop aging, there are ways to slow down the aging process and encourage healthy aging. These are a few methods:

1. **Healthy Diet:** Eating a balanced diet rich in fruits, vegetables, whole grains, lean proteins, and healthy fats can provide essential nutrients and antioxidants that help protect against cellular damage and support overall health [17].
2. **Regular Exercise:** Engaging in regular physical activity helps maintain muscle mass, bone density, and cardiovascular health. Exercise also reduces the risk of chronic diseases associated with aging, such as heart disease, diabetes, and osteoporosis.
3. **Adequate Sleep:** Getting enough high-quality sleep is crucial for cellular repair, cognitive function, and overall well-being. Aim for 7-9 hours of sleep per night and establish a consistent sleep schedule [18].
4. **Stress Management:** Chronic stress can accelerate aging by contributing to inflammation and oxidative stress. Practice stress-reduction techniques such as mindfulness meditation, deep breathing exercises, yoga, or spending time in nature.
5. **Sun Protection:** Protecting your skin from the sun's harmful UV rays can help prevent premature aging, such as wrinkles, age spots, and skin cancer. Use sunscreen with an SPF of 30 or higher, wear protective clothing, and seek shade during peak sun hours.
6. **Avoiding Harmful Substances:** Minimize exposure to tobacco smoke, excessive alcohol consumption, and other harmful substances that can accelerate aging and increase the risk of chronic diseases [19].

7. **Maintaining Social Connections:** Strong social relationships and a sense of community have been linked to better physical and mental health outcomes as people age. Stay connected with friends, family, and community groups to maintain social support and engagement [20].
8. **Cognitive Stimulation:** Keep your brain active and engaged by challenging yourself with puzzles, games, learning new skills, and staying intellectually curious. This can help preserve cognitive function and reduce the risk of age-related cognitive decline.
9. **Regular Health Check-ups:** Stay proactive about your health by scheduling regular check-ups with your healthcare provider. Addressing health issues early can prevent complications and promote overall well-being.
10. **Genetic Counseling and Testing:** Some individuals may have genetic predispositions to certain age-related diseases. Genetic counseling and testing can help identify these risks early, allowing for personalized preventive strategies and interventions [21].

Chapter 2

Literature Review

2.1 Skin anti-aging strategies

Aging of the skin is a complicated natural process that is impacted by both external and internal factors. Many anti-aging techniques have been created in recent years as to the belief that one of the primary variables indicating an individual's general "well-being" and opinion on "health" is their skin's appearance and state of health. This piece aims to examine the most effective anti-aging techniques now used by dermatologists, covering cosmological techniques, topical and systemic medicinal agents, invasive treatments, and preventative measures [22].

2.2 Skin Aging & Modern Age Anti-Aging Strategies

The skin is the largest organ in the body that is subjected to the external environment, and it ages due to both internal and external causes. Characteristics including wrinkles, elasticity loss, laxity, and a rough-textured aspect are indicative of aging skin. Along with phenotypic modifications to epidermal cells, extracellular matrix components like collagen and elastin also undergo functional and structural modifications as a result of aging. Fundamental aging results in a variety of histological, physiological, and biochemical alterations as well as structural alterations in the skin that are a normal byproduct of biological changes throughout time. Hormonal fluctuations, skin site variation, and genetics all have an impact on fundamental aging. It has delicate creases that define its appearance. Significant alterations occur in the various layers of the skin with regards to histology, physiology, and biochemistry. Human skin seems as a built combined from an engineering perspective. The stratum corneum, which presents the stiff, thin cover layer, is followed by the more flexible layers of feasible epidermis and the epidermis and then the much more flexible right next layer of subcutaneous white adipose tissue. When a multi-layer structure is subjected to strain, its stiffer layers exhibit mechanical instability, which essentially manifest as wrinkling. When the skin's mechanical strain beyond a certain threshold, these disorders manifest in a hierarchical manner. The way they appear is primarily influenced by the mechanical property mismatch among right next skin layers or among the skin and subcutaneous white adipose tissue, by the strength and thickness proportions of the adhesive among the layers, by the rigidity of their stretching and tensile characteristics, and by the amount of stress trapped in individual layers. The significance of elastic fibers in skin aging is explained by the fact that as skin ages, its capacity to bend

is gradually reduced. This results in a reduction of the skin's stability versus wrinkles of up to four times. Physicians, scientists, and other professionals who practice anti-aging medicine do so because they think that current medical and scientific advancements can reduce, stop, or even eliminate the physical aging happening to humans [23].

2.3 Current Trends in Anti-Aging Strategies

Every living thing gradually deteriorates and breaks breakdown as a consequence of a complex web of chemical cascades that contribute to the aging processes. This is an expected process that can be crippling and raises the risk of many illnesses. For an extended period, researchers from academia and industry have endeavored to hinder or even reversal the aging process with the aim of mitigating clinical stress, reinstating performance, and enhancing lifespan. Narrow experimental validation and a lack of rigorous design of studies have hampered the search for effective therapies, despite extensive research. In this overview, we examine the state of knowledge regarding the biological mechanisms behind aging and how this knowledge both facilitates and restricts the interpretation of data obtained from experimental models that employ these processes. We also go over a few specific treatment approaches that have shown promise in these model settings and may find application in clinical settings. Finally, we suggest a unified strategy that is required to thoroughly vet present and upcoming treatments and evaluate them directly toward successful treatments [24].

Chapter 3

Purpose of the study

3.1 Purpose of the study

This study aims to explore the scientific foundations and effectiveness of emerging anti-aging interventions to provide a comprehensive understanding of their potential benefits and limitations.

- By examining cutting-edge research and clinical trials, this study seeks to identify and evaluate novel anti-aging interventions.
- Central to this study is the exploration of the underlying mechanisms through which these anti-aging strategies exert their effects.
- This study critically evaluates the existing clinical evidence supporting the efficacy of emerging anti-aging interventions.
- Implications for Future Research and Clinical Practice.

Chapter 4

Methodology

4.1 Data collection procedure

Look for books, conference proceedings, peer-reviewed journals, and legal guidelines pertaining to "**Emerging Anti-Aging Strategies Scientific Basis and Efficacy**" To locate pertinent papers, reputable databases such as PubMed, Scopus, Google Scholar, and Research Gate have been utilized.

It has been starting work for this in January 2024.



Every effort has been made to gather as much information as possible by using search terms such as "Pathophysiology of Aging," "Prevention of Aging," "Management of Aging," and "Etiology of Aging". These terms were checked for potential exploits on educational bibliographic databases, web-based search engines, PubMed, Research Gate, Google Scholar, and Medline.



An academic evaluation leads the inspection; about 35 publications are examined for this evaluation.



In an effort to streamline the process, compiling all data from 2005 to 2023 and all work completed in Microsoft Word.



Each assessment paper that was created was reviewed, and it got even more scholarly.

The gathered information has finally been distilled.

4.2 Data analysis strategy

Data analysis is the methodical application of statistical and/or logical tools for describing and illustrating, condensing and summarizing, and evaluating data. Microsoft Excel was used to analyses the data.

Chapter 5

Results & Discussion

5.1 Autophagy enhancement for facilitating successful aging

Lysosomal disintegration, or autophagy, is the body's defensive housekeeping mechanism for getting rid of long-term Proteins that have been colonization infections, and ruined cell organelles. Through an action that breaks down intracellular components to maintain energy homeostasis, autophagy protects cells against stress. Development and illness are significantly influenced by autophagy. In instance, it has been proposed that increasing autophagy may help treat a number of illnesses, such as infectious diseases, malignancies, neurological ailments, and metabolic problems. Neuro-degeneration is also facilitated by defective autophagy [25]. Although autophagy aids in the removal of harmful protein types like tau in Alzheimer's disease and α -synuclein in Parkinson's disease, neurons with intact autophagy functions have a neuroprotective impact. Medication with autophagy promoters like rapamycin and lithium produced a number of beneficial effects in animal models of Parkinson's disease, including reduced α -synuclein accumulation, oxidative stress, mitochondrial dysfunction, and neuro-degeneration. One important factor in controlling the aging processes is autophagy. Many research utilizing yeasts, worms, flies, and mice have shown that increased expression of autophagy-related genes is a need for lifetime consequently, indicating the role of autophagy in aging [26]. Certain studies have also demonstrated that extending lifespan through tissue-specific expression of a single autophagy gene is sufficient, while additional investigations have highlighted the importance of distinct forms of autophagy in preventing aberrant accumulation of dysfunctional cellular components and concentrating on them. It's interesting to note that calorie restriction and food deprivation (CR) contribute to slower aging and improve longevity; this is made possible by upregulating autophagy, as CR's anti-aging effects are lessened when autophagy is suppressed. Furthermore, it has been demonstrated by previous research that improving autophagy may restore the ability of aged stem cells to regenerate [27].

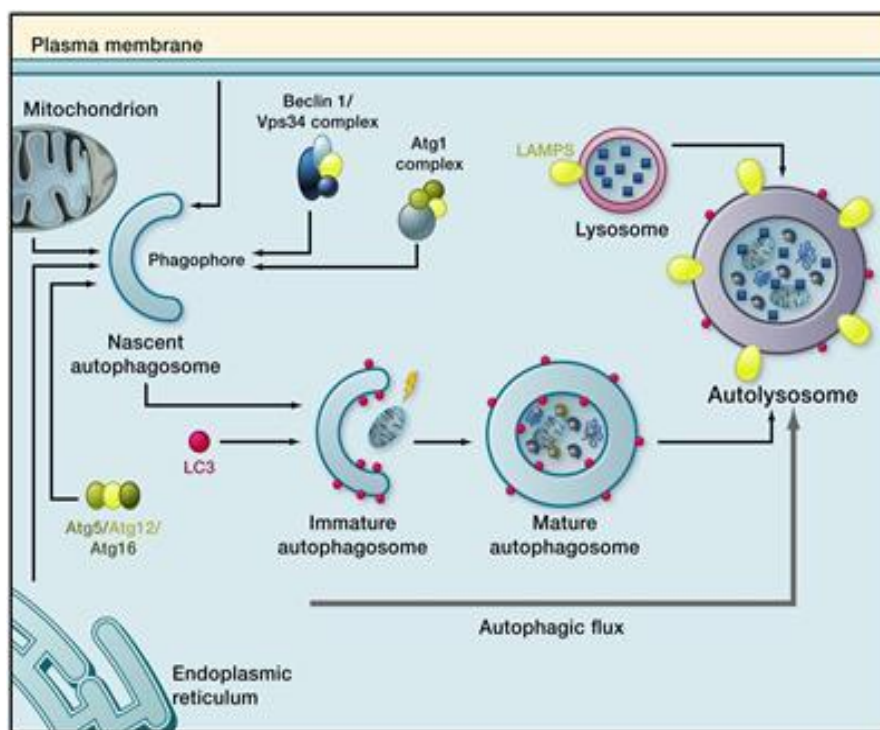


Figure 2: Influence of Normal Aging on Brain Autophagy

5.2 Senescent cell elimination as an anti-aging therapy

The role of senescent cells (SCs) between the agents of organismal aging has attracted a lot of attention. An irreversible cell-cycle arrest, resistance to apoptosis, and the development of a pro-inflammatory, tissue-destructive aging-associated secretion phenotype are characteristics of cellular senescence, which can be induced by many intracellular and extracellular stimuli. For example, cellular senescence may also be induced by a microbial infection. Senescent cells are unable to reproduce, which prevents them from taking part in normal tissue protection and reconstruction procedures. Instead, senescent cells produce a multitude of bioactive substances that obstruct regrowth and promote inflammation, which upsets the environment [28]. Senescent cells that express the cell cycle-inhibitory protein p16INK4A actively drive age-associated tissue degradation that results from aging, which in turn promotes a number of age-related illnesses. In fact, a variety of senescence-related indicators are present in patients with neurodegenerative disorders. Furthermore, loss of resiliency, metabolic instability, and stem cell failure can all be facilitated by the senescence-linked secretory phenotype. Therefore, simply by postponing the formation of senescent cells or reducing the impact of senescent cells, it

may be feasible to delay, avoid, or mitigate various problems linked with aging. Senescence is a typical aspect of aging in many types of tissues and organs, with a rise in senescent cells with advancing age [29]. Moreover, an investigation by Musi and associates has demonstrated that senescence of cells in the brain occurs concurrently with tau protein accumulation. Additional analysis confirmed that treatment with senolytic drugs led to decreased neurofibrillary tangle density, neuron loss, and increased ventricular size in tau transgenic mice with late-stage disease. These findings point to a connection among brain cellular aging and neurofibrillary tangles. An association among cognition-associated neuro-degeneration and the accumulation of senescent cells was proposed by another investigation. This work used a tau-dependent neurodegenerative illness model in which senescent microglia and astrocytes develop p16INK4A-positive. In these mice, the removal of aged astrocytes and microglia employing genetic methods stopped gliosis, hyperphosphorylation of soluble and insoluble tau, and neuronal degeneration in the cerebral cortex and the hippocampal regions [30]. It's amazing how these modifications, which were brought about by the removal of senescent cells, saved cognitive function. Furthermore, tau aggregation was likewise controlled by administration with a senior lytic medication.



Figure 3: Overview of various antiaging strategies

5.3 Intermittent fasting as a means to combat aging

It is evident through both epidemiology and experimental investigations that diet and eating patterns have a major role in the etiology of numerous age-related disorders. For instance, cutting calories by 20–40% under ad libitum is part of the most effective nongenetic dietary interventions (CR) that can lengthen life. According to an examination on adult dietary consumption, Okinawans ingested 17% fewer calories than the average adult in Japan and 40% less than the typical adult in the United States. Okinawa is a Japanese island home to nearly five times the world's population of centenarians. Numerous studies on animal prototypes have shown that a lower calorie consumption increases longevity [31]. Numerous research conducted subsequently have demonstrated that CR and intermittent fasting (IF) can have comparable benefits. Advantages of cardiovascular health include lowered blood cholesterol and body mass index, enhanced glucose tolerance, safeguarding the heart from ischemia injury, and a decreased risk of coronary artery disease. In pre-clinical research, IF was shown to have beneficial effects on brain health, including postponed onset of age-related brain deficits and enhanced cognitive performance with lower oxidative stress in middle age when IF was started in young adulthood. Moreover, IF reduced inflammasome activity, inhibited harmful genetic processes, enhanced recuperation, and altered forebrain neurogenesis, autophagy, and apoptosis in models of ischemic stroke [32]. In pre-clinical research, IF was shown to have beneficial effects on brain health, including postponed onset of age-related brain deficits and enhanced cognitive performance with lower oxidative stress in middle age when IF was started in young adulthood. Additionally, IF reduced inflammasome operation, inhibited harmful genetic processes, enhanced recuperation, and altered forebrain neurogenesis, autophagy, and apoptosis in models of ischemic stroke. In an animal model of widespread bacterial infection, lipopolysaccharide injection was proven to be an effective way for IF to reduce neuro inflammation and preserve cognitive function. Moreover, IF improved the psychological deficiencies seen in 3xTgAD animals. IF elevated β -hydroxybutyrate in the APP/PS1 model of AD mice, which subsequently suppressed the up-regulation of brain-derived lipoprotein lipase via down-regulating microRNA-29a [33].

Skin antiaging approach	Description
Cosmetological care	Daily skin care, Correct sun protection, Aesthetic non-invasive procedures, Chemical peelings, visible light devices, intense pulsed light (IPL), ablative and non-ablative laser photo-rejuvenation, radiofrequency (RF)
Topical medical agents or topical agents	Antioxidants, Cell regulators
Invasive procedures	Injectable skin bio-stimulation and rejuvenation, prevention of dynamic wrinkles, correction of static, anatomical wrinkles, restoration (redistribution) of fat and volume loss, skin augmentation and contouring, restoration (redistribution) of fat and volume loss, skin augmentation and contouring
Systemic agents	Hormone replacement therapy (HRT)
Avoiding of exogenous factors of aging, correction of life style and habits	Smoking, Pollution, Solar UV irradiation. Stress, Nutrition, diet restriction and alimentary supplementation, Physical activity, Control of general health
Preventive medicine	

Table 1: Skin antiaging approach

5.3.1 Satisfaction level of the consumer with antiaging product used

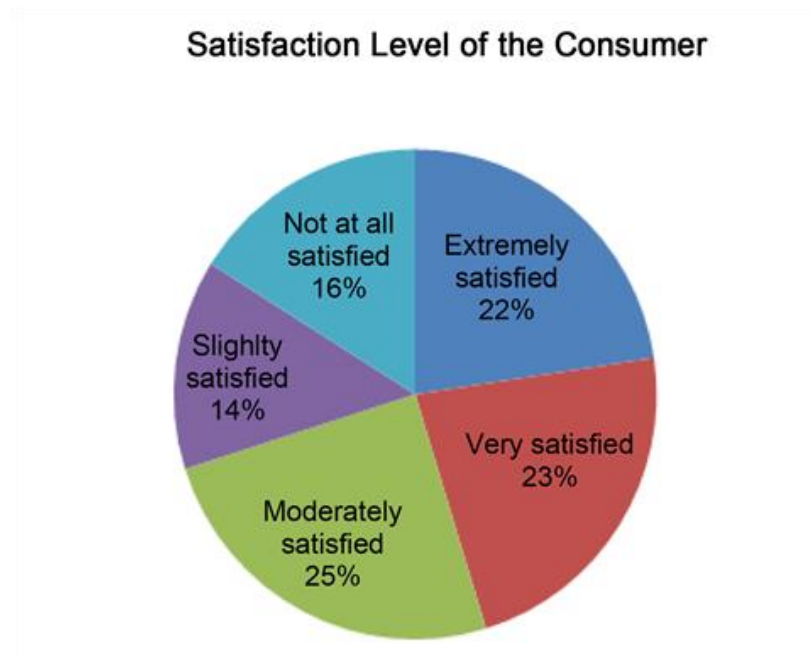


Figure 4: Satisfaction level of the consumer with antiaging product used

The majority of women concurred that they place a great deal of importance on their skin. As can be seen, 23% of respondents appear to be happy with the product, while 16% do not think it is beneficial at all.

5.3.2 Duration of use of the participants of antiaging cosmetics

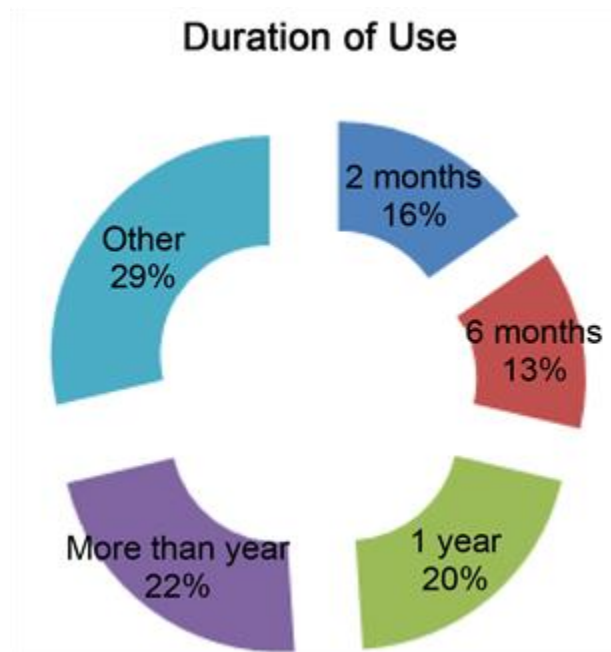


Figure 5: Duration of use of the participants of antiaging cosmetics

Only 22 candidates had been taking antiaging products for the previous eight weeks, 19 participants for the previous six months, 29 for one year, 32 for more than a year, and 41 participants for even longer more than a year.

5.4 Photo protection and Systemic Antioxidants

Photo aging, also known as extrinsic skin aging, is a symptom of chronic damage from sunlight of the skin. Reactive oxygen species (ROS) produced by UV light and DNA damage from sunlight are the first molecular processes that cause the majority of the common histologic and clinical signs of chronic skin photodamage. The most significant cutaneous symptoms of precocious photoaging are thought to be wrinkles and pigmentary alterations. Sun evasion, sun protection with sun protection to block or minimize UV radiation exposure to the skin, retinoids to suppress the synthesis of collagenase and increase the creation of collagen, and antioxidants, especially when combined, to lower and eliminate free radicals (FR) are some of the techniques used for avoiding photo-aging [34]. According to interventional research, administering specific nutritional supplements can actually enhance skin problems and postpone the aging process of the skin. The primary function of nutritional antioxidants is to gather free radicals (FRs). They do this in

four ways: (1) by immediately. Both non-enzymatic (such as uric acid, glutathione, bilirubin, thiols, albumin, and nutritional components, such as vitamins and phenols) and mechanical (such as superoxide breaks down, glutathione peroxidases (GSHPx), and catalase) physiological antioxidant defenses exist [35].

Available antioxidant For anti-aging	Mechanism	Reference
Vitamin C (Ascorbic Acid)	Acts as a free radical scavenger, neutralizing reactive oxygen species (ROS) and preventing oxidative damage to proteins, lipids, and DNA. Additionally, it regenerates other antioxidants like vitamin E, enhancing the overall antioxidant defense system.	[23]
Vitamin E (Alpha-Tocopherol)	Protects cell membranes from oxidative damage by scavenging lipid peroxyl radicals, thus preventing lipid peroxidation. It also works synergistically with vitamin C and other antioxidants.	
Glutathione	Acts as a major intracellular antioxidant, directly neutralizing ROS and reactive nitrogen species (RNS). It also plays a crucial role in detoxification processes by conjugating with toxins, thereby protecting cells from damage.	[24]
Coenzyme Q10 (Ubiquinone)	Essential for mitochondrial function and energy production. It acts as an antioxidant by scavenging free radicals, particularly within the mitochondria where ROS generation is high, thus protecting cells from oxidative damage and improving overall cellular function.	
Resveratrol	Functions as a potent antioxidant and activates sit-ins, which are enzymes associated with longevity and cellular health. Resveratrol also modulates various signaling pathways involved in inflammation, apoptosis, and cell survival.	[25]
Curcumin (from Turmeric)	Exhibits antioxidant properties by scavenging free radicals and enhancing the activity of antioxidant enzymes. It also possesses anti-inflammatory effects by inhibiting NF-κB and other pro-inflammatory pathways implicated in aging processes.	
Polyphenols (e.g., from green tea, berries, and dark chocolate)	Act as antioxidants by scavenging free radicals and chelating transition metal ions involved in ROS generation. Polyphenols also exert anti-inflammatory effects and modulate signaling pathways associated with aging and age-related diseases.	[26]

Astaxanthin	A carotenoid pigment known for its potent antioxidant properties, particularly in scavenging singlet oxygen and quenching lipid peroxy radicals.	
-------------	--	--

Table 2: Available antioxidant for anti-aging with mechanism

5.5 Topical Pharmacological Agents with Anti-Aging Properties

Antioxidants and cell controllers are the two main classes of chemicals that can be included in antiaging creams. By lowering the amount of FR in the tissues, antioxidants like vitamins, polyphenols, and flavonoids slow down the deterioration of collagen. Growth factors (GF), retinols, and peptides are examples of cell controllers that directly affect collagen metabolism and have an impact on collagen formation.

The most significant antioxidants are vitamins C, B3, and E because of their low molecular size, which allows them to permeate the skin.⁵² It has been demonstrated that water-soluble, heat-labile local L-ascorbic acid (vitamin C) at rates ranging from 5–15% has an anti-aging impact on cutaneous by promoting the development of Col-1, Col-3, and enzymes crucial for collagen synthesis [36]. Research has demonstrated that the antioxidative defense provided by a mixture of vitamins C and E is greater than that of either vitamin alone. Vitamin B3, niacinamide, controls the metabolism of cells and renewal and is utilized as an antiaging treatment at a concentration of 5%.⁵⁶ After three months of topical therapy, improvements in skin elasticity, erythema, and pigmentation have been noted in certain investigations. When added to skin products, vitamin E (α -tocopherol) exhibits anti-inflammatory and antiproliferative actions at doses ranging from 2 to 20%. It works by straightening the skin, boosting the stratum corneum's capacity to retain humidity, quickening the process of epithelialization, and enhancing face protection from sunlight. The results aren't as potent as those of vitamins C [37].

5.6 Stem cell therapy for promoting healthy brain aging and reversing AD

In order to enhance the activity of the brain as it ages, the effectiveness of intracerebral transplanting or peripheral injection of various stem cells, such as mesenchymal stem cells (MSCs), neural stem cells (NSCs), or glial-restricted progenitors (GRPs), has been investigated in animal models. Hattiangady et al.'s work showed that transplanting NSCs

or GRPs into the aged hippocampal region stimulates native NSCs in the sub-granular zone and increases the generation of new dentate granule cells. This work offered the first evidence that transplanting GRPs or NSCs is a promising method for enhancing neurogenesis in the aged hippocampal regions [38]. All of these findings pointed to the possibility that NSC transplantation, by stimulating endogenous neurogenesis, can encourage repair in the aging brain. Shetty and Hattiangady's over time investigation also showed that the aged hippocampal may support strong NSC graft-derived cell integration and development. The capacity of transplanted NSCs to create neurogenic niches in non-neurogenic areas of the aged hippocampal tissue is an intriguing discovery. Even three months after grafting, the emergence of new, immature neurons from graft-derived cells inside graft cores situated in the non-neurogenic regions demonstrated the existence of new neurogenic niches [39]. It was further shown by sequential labelling with several birth-dating indicators that the juvenile neurons discovered inside graft cores were, in fact, produced by graft-derived cells in the elderly hippocampal region. As freshly created neurons and glia are predicted to enhance not only the microenvironment but also the plasticity and function of the aged hippocampus, this occurrence is advantageous if these niches can continually generate new neurons and glia in the transplanted hippocampus. Overall, these findings are important since older people would primarily be the beneficiaries of any prospective NSC grafting applications for the management of neurodegenerative disorders in the early phases of the disease's development and age-related deficits [40]. Benefits from treatment with stem cells have been demonstrated in multiple additional age-related neurological disease models. The memory and spatial learning deficiencies in aged 3xTg-AD mice were restored by hippocampus NSC transplantation, according to a study by Blurton-Jones and colleagues. There was an improvement in cognitive performance without a change in AD pathology. Investigations on both gain-of-function and loss-of-function participants found that increased hippocampal synaptic density, mediated by BDNF, was the process driving enhanced cognitive function.

Chapter 6

Conclusion

6.1 Conclusion

Numerous anti-aging techniques are being developed which have demonstrated significant promise in reducing the rate of aging or postponing the onset of illnesses associated with aging. Multiple preliminary research findings suggest that the following strategies hold promise for maintaining normal health throughout aging and delaying age-related illnesses like AD: upregulating autophagy by means of autophagy enhancements, eliminating senescent cells with senolytics, blood transfusion of young blood plasma, neurogenesis, and BDNF improvement through specific drugs. Nevertheless, in order to ascertain these methods' long-term effectiveness and absence of detrimental effects on the functioning of diverse tissues and organs, they will need to be critically evaluated in clinical studies. On the other hand, because they are non-intrusive and appear to have few side effects, strategies like various forms of IF, routine PE, RESV, and CUR therapy are prepared for extensive clinical trials. The use of stem cells, specifically NSCs, GRPs, and MSCs, has demonstrated potential in promoting restoration and enhancing brain function in the elderly or AD population. However, in order to further develop stem cell therapy to overcome brain aging or for reversing the pathology and symptoms of AD, efficacy and safety trials involving cells derived from hiPSCs or hESCs would be necessary. These sources can supply an infinite number of specific cell types required for cell treatment in different diseases.

Chapter 7

Reference

References

- [1] Gurau F, Baldoni S, Prattichizzo F, Espinosa E, Amenta F, Procopio AD, et al. (2018). Anti-senescence compounds: A potential nutraceutical approach to healthy aging. *Ageing Res Rev*, 46:14-31.
- [2] Aunan JR, Cho WC, Soreide K (2017). The Biology of Aging and Cancer: A Brief Overview of Shared and Divergent Molecular Hallmarks. *Aging Dis*, 8:628-642.
- [3] de Magalhaes JP, Stevens M, Thornton D (2017). The Business of Anti-Aging Science. *Trends Biotechnol*, 35:1062-1073.
- [4] Sun N, Youle RJ, Finkel T (2016). The Mitochondrial Basis of Aging. *Mol Cell*, 61:654-666.
- [5] Szybinska A, Lesniak W (2017). P53 Dysfunction in Neurodegenerative Diseases - The Cause or Effect of Pathological Changes? *Aging Dis*, 8:506-518.
- [6] Xu Z, Feng W, Shen Q, Yu N, Yu K, Wang S, et al. (2017). Rhizoma Coptidis and Berberine as a Natural Drug to Combat Aging and Aging-Related Diseases via Anti-Oxidation and AMPK Activation. *Aging Dis*, 8:760-777.
- [7] Zamroziewicz MK, Paul EJ, Zwillling CE, Barbey AK (2017). Predictors of Memory in Healthy Aging: Polyunsaturated Fatty Acid Balance and Fornix White Matter Integrity. *Aging Dis*, 8:372-383.
- [8] Zinger A, Cho WC, Ben-Yehuda A (2017). Cancer and Aging - the Inflammatory Connection. *Aging Dis*, 8:611-627.
- [9] Stambler I (2017). Recognizing Degenerative Aging as a Treatable Medical Condition: Methodology and Policy. *Aging Dis*, 8:583-589.
- [10] Chakrabarti S, Mohanakumar KP (2016). Aging and Neurodegeneration: A Tangle of Models and Mechanisms. *Aging Dis*, 7:111-113.
- [11] Konar A, Singh P, Thakur MK (2016). Age-associated Cognitive Decline: Insights into Molecular Switches and Recovery Avenues. *Aging Dis*, 7:121-129.

- [12] de Magalhaes JP (2012). Programmatic features of aging originating in development: aging mechanisms beyond molecular damage? *FASEB J*, 26:4821-4826.
- [13] Kenyon CJ (2010). The genetics of ageing. *Nature*, 464:504-512.
- [14] Tacutu R, Craig T, Budovsky A, Wuttke D, Lehmann G, Taranukha D, et al. (2013). Human Ageing Genomic Resources: integrated databases and tools for the biology and genetics of ageing. *Nucleic Acids Res*, 41:D1027-1033.
- [15] Bagherniya M, Butler AE, Barreto GE, Sahebkar A (2018). The effect of fasting or calorie restriction on autophagy induction: A review of the literature. *Ageing Res Rev*, 47:183-197.
- [16] Redmann M, Darley-Usmar V, Zhang J (2016). The Role of Autophagy, Mitophagy and Lysosomal Functions in Modulating Bioenergetics and Survival in the Context of Redox and Proteotoxic Damage: Implications for Neurodegenerative Diseases. *Aging Dis*, 7:150-162.
- [17] Hansen M, Rubinsztein DC, Walker DW (2018). Autophagy as a promoter of longevity: insights from model organisms. *Nat Rev Mol Cell Biol*, 19:579-593.
- [18] Scrivo A, Bourdenx M, Pampliega O, Cuervo AM (2018). Selective autophagy as a potential therapeutic target for neurodegenerative disorders. *Lancet Neurol*, 17:802-815.
- [19] Moors TE, Hoozemans JJ, Ingrassia A, Beccari T, Parnetti L, Chartier-Harlin MC, et al. (2017). Therapeutic potential of autophagy-enhancing agents in Parkinson's disease. *Mol Neurodegener*, 12:11. [20] Li Y, Wang K, Zhou K, Guo W, Dai B, Liang Y, et al. (2018). Novel D-A-D based near-infrared probes for the detection of beta-amyloid and Tau fibrils in Alzheimer's disease. *Chem Commun (Camb)*, 54:8717-8720.
- [21] Hamano T, Hayashi K, Shirafuji N, Nakamoto Y (2018). The Implications of Autophagy in Alzheimer's Disease. *Curr Alzheimer Res*, 15:1283-1296.
- [22] Arensman MD, Eng CH (2018). Self-Digestion for Lifespan Extension: Enhanced Autophagy Delays Aging. *Mol Cell*, 71:485-486.

- [23] Fernandez AF, Sebti S, Wei Y, Zou Z, Shi M, McMillan KL, et al. (2018). Disruption of the beclin 1-BCL2 autophagy regulatory complex promotes longevity in mice. *Nature*, 558:136-140.
- [24] Shamalnasab M, Gravel SP, St-Pierre J, Breton L, Jager S, Aguilaniu H (2018). A salicylic acid derivative extends the lifespan of *Caenorhabditis elegans* by activating autophagy and the mitochondrial unfolded protein response. *Aging Cell*:e12830.
- [25] Gelino S, Chang JT, Kumsta C, She X, Davis A, Nguyen C, et al. (2016). Intestinal Autophagy Improves Healthspan and Longevity in *C. elegans* during Dietary Restriction. *PLoS Genet*, 12:e1006135.
- [26] Madeo F, Zimmermann A, Maiuri MC, Kroemer G (2015). Essential role for autophagy in life span extension. *J Clin Invest*, 125:85-93.
- [27] Shakeri A, Cicero AFG, Panahi Y, Mohajeri M, Sahebkar A (2018). Curcumin: A naturally occurring autophagy modulator. *J Cell Physiol*. Sep 21, [Epub ahead of print].
- [28] Revuelta M, Matheu A (2017). Autophagy in stem cell aging. *Aging Cell*, 16:912-915.
- [29] Chung JH, Manganiello V, Dyck JR (2012). Resveratrol as a calorie restriction mimetic: therapeutic implications. *Trends Cell Biol*, 22:546-554.
- [30] Kodali M, Parihar VK, Hattiangady B, Mishra V, Shuai B, Shetty AK (2015). Resveratrol prevents age related memory and mood dysfunction with increased hippocampal neurogenesis and microvasculature, and reduced glial activation. *Sci Rep*, 5:8075.
- [31] Morselli E, Galluzzi L, Kepp O, Criollo A, Maiuri MC, Tavernarakis N, et al. (2009). Autophagy mediates pharmacological lifespan extension by spermidine and resveratrol. *Aging (Albany NY)*, 1:961-970.
- [32] Shetty AK (2011). Promise of resveratrol for easing status epilepticus and epilepsy. *Pharmacol Ther*, 131:269-286.

- [33] Eisenberg T, Abdellatif M, Schroeder S, Primessnig U, Stekovic S, Pendl T, et al. (2016). Cardioprotection and lifespan extension by the natural polyamine spermidine. *Nat Med*, 22:1428-1438.
- [34] Madeo F, Carmona-Gutierrez D, Kepp O, Kroemer G (2018). Spermidine delays aging in humans. *Aging (Albany NY)*, 10:2209-2211.
- [35] Ren J, Zhang Y (2018). Targeting Autophagy in Aging and Aging-Related Cardiovascular Diseases. *Trends Pharmacol Sci*, 39:1064-1076.
- [36] Zhang L, Hu T, Amombo E, Wang G, Xie Y, Fu J (2017). The Alleviation of Heat Damage to Photosystem II and Enzymatic Antioxidants by Exogenous Spermidine in Tall Fescue. *Front Plant Sci*, 8:1747.
- [37] Bussian TJ, Aziz A, Meyer CF, Swenson BL, van Deursen JM, Baker DJ (2018). Clearance of senescent glial cells prevents tau-dependent pathology and cognitive decline. *Nature*, 562:578-582.
- [38] Kirkland JL, Tchkonian T (2017). Cellular Senescence: A Translational Perspective. *EBioMedicine*, 21:21-28.
- [39] Sun H, Paixao L, Oliva JT, Goparaju B, Carvalho DZ, van Leeuwen KG, et al. (2018). Brain age from the electroencephalogram of sleep. *Neurobiol Aging*, 74:112-120.
- [40] Wei W, Ji S (2018). Cellular senescence: Molecular mechanisms and pathogenicity. *J Cell Physiol*, 233:9121-9135.