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Natural Senolytics: A New-Generation Nutraceutical approach for Health Rejuvenation

Dr. Vikrant Narwal[1], Charu Kamboj[2] and Dr. Sonika Choudhary[3,4]*
[1]Director, Astonea Labs Pvt. Ltd., Panchkula, Haryana, India
Email: vikrantnarwal7@gmail.com
[2]Sr. Manager, Scientific Content Coordinator, Astonea Labs Pvt. Ltd., Panchkula, Haryana, India
Email: kambojcharu1997@gmail.com
[3]Assistant Professor (Former), Department of Dairy Chemistry, College of Dairy and Food Science Technology, Guru Angad Dev Veterinary and Animal Sciences University, Ludhiana, Punjab, India
Email: sonikachoudhary15@gmail.com
[4]Chief Quality Assurance, Astonea Labs Pvt. Ltd., Panchkula, Haryana, India
*Corresponding Author: sonikachoudhary15@gmail.com
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Abstract

Senolytics represent a promising therapeutic approach that targets the fundamental pillars of cellular dysfunction rather than individual diseases. These agents selectively eliminate senescent or "zombie" cells, accumulated over the time and release pro-inflammatory factors known as the Senescence-Associated Secretory Phenotype (SASP). This chronic inflammatory environment contributes to tissue damage, metabolic imbalance, and increased disease risk. By disrupting senescent cell anti-apoptotic pathways (SCAPs), senolytics enable these damaged cells to undergo programmed cell death while sparing healthy cells. Complementing this approach, senomorphics suppress the harmful secretions of senescent cells without removing them. Natural compounds such as quercetin, fisetin, curcumin, and resveratrol have emerged as safer alternatives to synthetic drugs, acting through pathways linked to inflammation control and cellular repair. An important advancement is the integration of senolytics with the CD38/NAD+/SIRT1 axis. Elevated CD38 activity depletes NAD+, impairing cellular energy and repair mechanisms. Senolytics help preserve NAD+ levels and enhance SIRT1 activity, particularly when combined with NAD+ precursors and mitochondrial support like CoQ10. Together, this integrated strategy promotes reduced inflammation, improved metabolic function, and enhanced cellular resilience, supporting healthier lifespan extension.

1. Introduction

Aging is a progressive biological process characterized by a decline in physiological integrity, leading to impaired function and increased vulnerability to chronic diseases. Old age

is linked to a number of deleterious conditions, such as cancer, neurodegenerative dieases, cardiovascular diseases, and metabolic disorders. As per Geroscience (An interdisciplinary field studying the biological mechanisms of aging to prevent, delay, or treat multiple chronic age-related diseases simultaneously, rather than individually) targeting fundamental aging mechanisms, rather than individual diseases, offers profound health and financial benefits. Cellular senescence or cell death is a state of cell in which cell becomes dead or unresponsive owing to various factors like DNA damage, telomere shortening, or oncogenic stimuli. While cellular senescence plays significant role in tissue repair and tumor suppression, the chronic accumulation of these senescent cells (SCs) or zombie cells creates a pro-inflammatory environment through the Senescence-Associated Secretory Phenotype (SASP). This phenotype involves the secretion of various chemical messengers like cytokines (e.g., IL-6), chemokines, and proteases that can cause serious damage to surrounding tissues and promote systemic inflammation in body. To overcome these harmful effects, two primary senotherapeutic strategies have emerged: senolytics and senomorphics. Senolytics are drugs that selectively eliminate senescent cells by temporarily disabling the anti-apoptotic pathways (SCAPs) that allow them to persist. In contrast, senomorphics aim to suppress or modulate the harmful aspects of the SASP without killing the cells. In addition to this there are certain natural senolytics derived from dietary phytochemicals which offers a promising, low-toxicity alternative to synthetic compounds. Bioactive molecules such as quercetin (found in onions and apples), fisetin (in strawberries), and curcumin (in turmeric) have demonstrated the ability to selectively target SCs or dampen the SASP in preclinical models. These compounds often work by activating protective pathways like SIRT1, which regulates DNA repair and metabolism. Complementing these strategies is the restoration of nicotinamide adenine dinucleotide (NAD+) levels, which naturally decline with age. Supplementing senolytics with NAD+ precursors, such as nicotinamide mononucleotide (NMN) or nicotinamide riboside (NR), can revitalize mitochondrial function and enhance the efficacy of metabolic functions during aging. Together, the integration of natural senolytics and NAD+ boosters represents a multilevel strategy for multi-systemic heath rejuvenation, aiming to extend healthy life span and reduce the burden of age-related disease in humans.

2. Types of Senotherapies

Senotherapeutics targets a diverse range of pharmacological strategies aimed at mitigating the deleterious effects of cellular senescence on tissue homeostasis and healthspan. These therapies are primarily categorized into three groups based on their molecular approach: senolytics, senomorphics, and the emerging field of senoreversal.

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Senolytics are agents designed to selectively eliminate senescent cells by inducing apoptosis. Senescent cells are characterized by their resistance to programmed cell death, which they achieve by upregulating pro-survival networks known as senescent cell anti-apoptotic pathways (SCAPs). Senolytic drugs, such as Dasatinib, Quercetin, and Navitoclax, work by temporarily disabling these SCAPs, thereby allowing the tissue-destructive senescent cells to undergo apoptosis without harming neighboring healthy cells.

Senomorphics, Unlike senolytics, senomorphics do not kill the senescent cell; instead, they modulate its phenotype to resemble that of a "younger" cell. Their primary function is to suppress the senescence-associated secretory phenotype (SASP)—the cocktail of pro-inflammatory cytokines, chemokines, and proteases that contributes to chronic "inflammaging". Senomorphics target various signaling cascades, including mechanistic Target of Rapamycin (mTOR), Nuclear Factor kappa of activated B cells (NF-κB), and Mitogen-Activated Protein Kinases (MAPKs), to reduce the harmful paracrine effects of senescent cells while allowing the cells themselves to remain in a state of growth arrest. These three are pro-inflammatory signalling hub of body.

Senoreversal, A newer and more experimental approach is senoreversal, which seeks to functionally "reprogram" senescent cells back to their original, healthy state. While still in its infancy compared to the other two strategies, this approach represents a potential third pillar of senotherapy that could one day move beyond cell elimination or suppression toward total cellular restoration.

3. History and Generations of Senolytics

The field of senotherapy has evolved rapidly over the last decade, transitioning from basic cellular observations to sophisticated drug discovery programs. The development of senolytics—agents that selectively induce apoptosis in senescent cells (SCs)—can be categorized into distinct generations based on their discovery methods, molecular targets, and therapeutic focus. Apoptosis is your body's way of getting rid of damaged cells through a process called Programmed cell death. "Programmed" means your body knows these cells need to die and manages the process from start to finish. Instead of growing and dividing like healthy cells, cells that go through apoptosis follow a set of steps that lead to their breakdown.

The conceptual foundation of senolytics dates back to the discovery of the "Hayflick limit" in 1961, which identified that cells have a finite capacity for division. However, it was not until the early 21st century that researchers recognized that the chronic accumulation of these non-dividing cells contributes to "inflammaging" via the SASP. The first definitive

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"senolytic" drugs were identified in 2015, marking a shift from merely observing senescence to actively targeting it for therapeutic benefit (Fig.1)

First-Generation Senolytic

The first generation of senolytics was identified through mechanism-based discovery, specifically by targeting Senescent Cell Anti-Apoptotic Pathways (SCAPs). Researchers hypothesized that SCs survive in toxic inflammatory environments by upregulating specific pro-survival networks.

- Dasatinib and Quercetin (D+Q): The first identified senolytic combination. Dasatinib, a tyrosine kinase inhibitor, targets the Src family and other kinases, while Quercetin, a natural flavonoid, inhibits PI3K and other pro-survival pathways. This combination was found to be effective across a broader range of cell types than either agent alone.
- Navitoclax (ABT-263): A potent inhibitor of the Bcl-2 family (specifically Bcl-2, Bcl-xL, and Bcl-w). While highly effective in inducing apoptosis in senescent fibroblasts and endothelial cells, its clinical use is limited by side effects like thrombocytopenia (platelet reduction).

Second-Generation Senolytics

The second generation emerged through High-Throughput Screening (HTS) of compound libraries and more refined biological engineering.

- Specific Bcl-2 Inhibitors: To avoid the toxicity of Navitoclax, researchers developed more selective inhibitors like ABT-199, though these often-showed reduced efficacy across diverse SC types.
- Targeted Delivery Systems: This generation introduced toxin-loaded nanoparticles and Proteolysis-Targeting Chimeras (PROTACs), which selectively degrade pro-survival proteins like Bcl-xL specifically within SCs to reduce systemic toxicity.

New Generation: Natural Phytochemicals and Senomorphics

Current article emphasizes on sustainability Natural Phytochemicals by exploring their Natural Senolytics (phytochemicals) and Senomorphics activities.

- Natural Senolytics: Compounds like Fisetin and Piperlongumine have gained prominence for their ability to clear SCs with lower toxicity profiles than synthetic drugs. Fisetin is particularly noted for its potential to extend healthspan and alleviate frailty in preclinical models.
- Senomorphics: Rather than killing the cell, these agents (e.g., Metformin, Rapamycin) modulate the SASP to suppress chronic inflammation. This approach is ideal when SC clearance is undesirable or in tissues where cell replacement is difficult.

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Today, senolytics have progressed into numerous clinical trials for conditions ranging from Alzheimer's disease to idiopathic pulmonary fibrosis, representing a new frontier in geroscience.

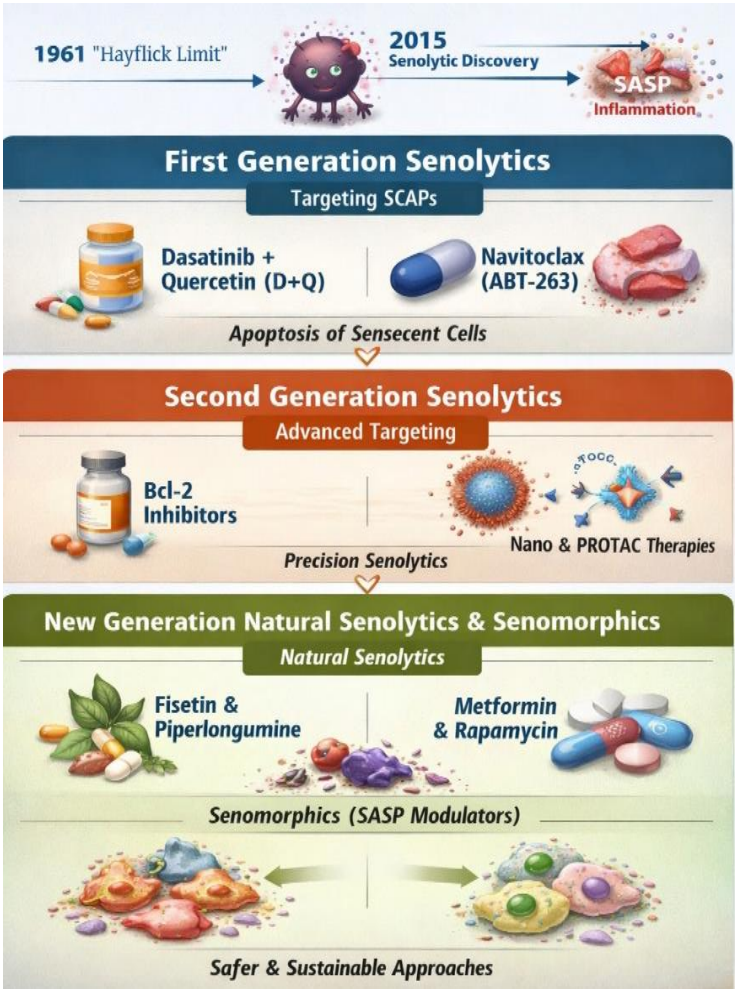

Fig. 1 : Generations of Senolytics

4. Natural Senolytic and Senomorphic Compounds: Bioactive Sources and Mechanisms

Natural senotherapeutics offer a sustainable, multi-targeted approach to clearing senescent cells (senolytics) or modulating their inflammatory secretions (senomorphics). These dietary phytochemicals are primarily categorized by their chemical structures, each possessing unique biological mechanisms that contribute to systemic rejuvenation.

Flavonoids and Polyphenols

Flavonoids are the most extensively studied natural senolytics, functioning primarily by temporarily disabling Senescent Cell Anti-Apoptotic Pathways (SCAPs). Fisetin mainly found abundantly in strawberries, apples, persimmons, and onions, fisetin is recognized as one of the most potent natural senolytics. It selectively induces apoptosis in senescent cells by inhibiting pro-survival pathways like PI3K/AKT/mTOR.

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- **Quercetin** derived from red onions, capers, kale, and apples, is a classic senolytic often used in combination with other agents (like Dasatinib) to broaden its effectiveness across different cell types. It works by inhibiting specific kinases and anti-apoptotic genes, such as PI3K.
- **Curcumin**, derived from turmeric (*Curcuma longa*), curcumin and its high-potency analogs (e.g., EF24) act as senomorphics by suppressing NF-κB signaling, which is the primary driver of the pro-inflammatory SASP.
- **Luteolin/Luteolinidin:** While Luteolin (from celery and herbs) serves as a master senomorphic by inhibiting the NF-κB/mTOR axis, its 3-deoxy counterpart, Luteolinidin (found in sorghum), is valued for its unique chemical stability and potential in modulating CD38/NAD+ metabolism.
- **Apigenin,** derived from parsley and chamomile plants, It can reduce COX2, IL6, and TNF-_ in response to bacterial toxins and lipopolysaccharide. Apigenin reduces the expression of COX2 and can also produce anti-inflammatory effects in the context of cardiovascular disease, neurodegeneration, diabetes, and cancer.

Carotenoids: Astaxanthin (found in Haematococcus pluvialis microalgae, wild salmon, and krill offers an array of health benefits by mitigating reactive oxygen species (ROS) and supporting mitochondrial. Lycopene **(tomatoes**, watermelon, and pink grapefruit) activates SIRT1 to impart muscle angiogenesis and reverse insulin. resistance in integrity

Stilbenes: Resveratrol and Pterostilbene

Stilbenes are non-flavonoid phenolic compounds that are critical to "geroprotective" nutrition due to their ability to activate longevity-linked pathways. **Resveratro**l is primarily found in the skin of red grapes, red wine, and berries, resveratrol is a well-known activator of SIRT1, an enzyme that promotes DNA repair and metabolic health. While it has lower bioavailability, it serves as a powerful senomorphic by dampening oxidative stress and NF-κB-mediated inflammation.

Pterostilbene found in blueberries and grapes, pterostilbene is a dimethylated analog of resveratrol. It is often preferred in supplement formulations because it exhibits 80% bioavailability compared to resveratrol's 20%. It enhances longevity-associated genes like SIRT2 and upregulates endogenous antioxidant enzymes such as superoxide dismutase (SOD).

Betaine (Trimethylglycine), Found in beets, spinach, wheat bran, and shellfish. Keeps protein stability and shields cells from stress, supporting healthy aging. Acts as Osmo protectant and anti-inflammatory agent.

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5. Nutri-Senolytic Coupling: CD38/NAD+/SIRT1 Axis

In the context of multi-systemic health rejuvenation, the CD38/NAD+/SIRT1 axis is a fundamental metabolic framework that regulates how our cells age and repair themselves. Senolytics play a crucial role in this axis by removing the primary obstacles to its efficient functioning.

The CD38/NAD+/SIRT1 axis acts as a biological thermostat for cellular energy and repair. Its three main components work in a delicate balance:

- NAD+ (Nicotinamide Adenine Dinucleotide): This is a critical coenzyme present in all living cells, essential for energy metabolism and DNA repair. However, NAD+ levels decline sharply as we age.
- CD38: This is an enzyme that acts as a primary "consumer" or degrader of NAD+. During aging, CD38 levels increase—largely driven by the inflammatory secretions of senescent cells—which leads to the depletion of the body's NAD+ stores.
- SIRT1 (Sirtuin 1): Known as a "longevity protein," SIRT1 is an enzyme that depends on NAD+ to function. It promotes DNA repair, mitochondrial health, and metabolic stability. When NAD+ is low (due to high CD38), SIRT1 becomes inactive, leading to accelerated cellular aging.

How Senolytics Interact with the Axis: Senolytics provide synergy by addressing the root cause of the axis's breakdown:

1. Reducing CD38 Activity: Senescent cells are a major source of the inflammation that triggers high CD38 levels. By selectively eliminating these cells, senolytics indirectly reduce CD38, thereby "saving" NAD+ from degradation.
2. Enabling SIRT1: By helping to restore the NAD+ pool, senolytics ensure that enzymes like SIRT1 have the fuel they need to perform critical repair functions.
3. Senolytics that Provide Synergy with NAD+: Certain natural senolytics are particularly effective at boosting the benefits of NAD+ precursors (like NMN or NR) through direct and indirect mechanisms:

Apigenin is a potent inhibitor of CD38. By blocking this enzyme, it prevents the destruction of NAD+, making it one of the most direct synergistic partners for NAD+ boosting supplements. Quercetin is often used alongside NAD+ precursors. It helps by inhibiting the pro-inflammatory pathways that lead to NAD+ depletion and has been shown to improve metabolic health markers. Fisetin mimics some of the effects of NAD+ by activating SIRT1 directly. It induces mitochondrial fragmentation in a way that increases the NAD+/NADH ratio, essentially "priming" the cell for rejuvenation.

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Resveratrol is a classic SIRT1 activator. When taken with NAD+ precursors, it ensures that the newly available NAD+ is immediately used by sirtuins for cellular repair.

By combining these senolytics with NAD+ precursors, a " **biochemical coupling** " is formed, senolytics remove the cells that drain NAD+, while precursors refill the tank, allowing SIRT1 to restore youthful cellular function.

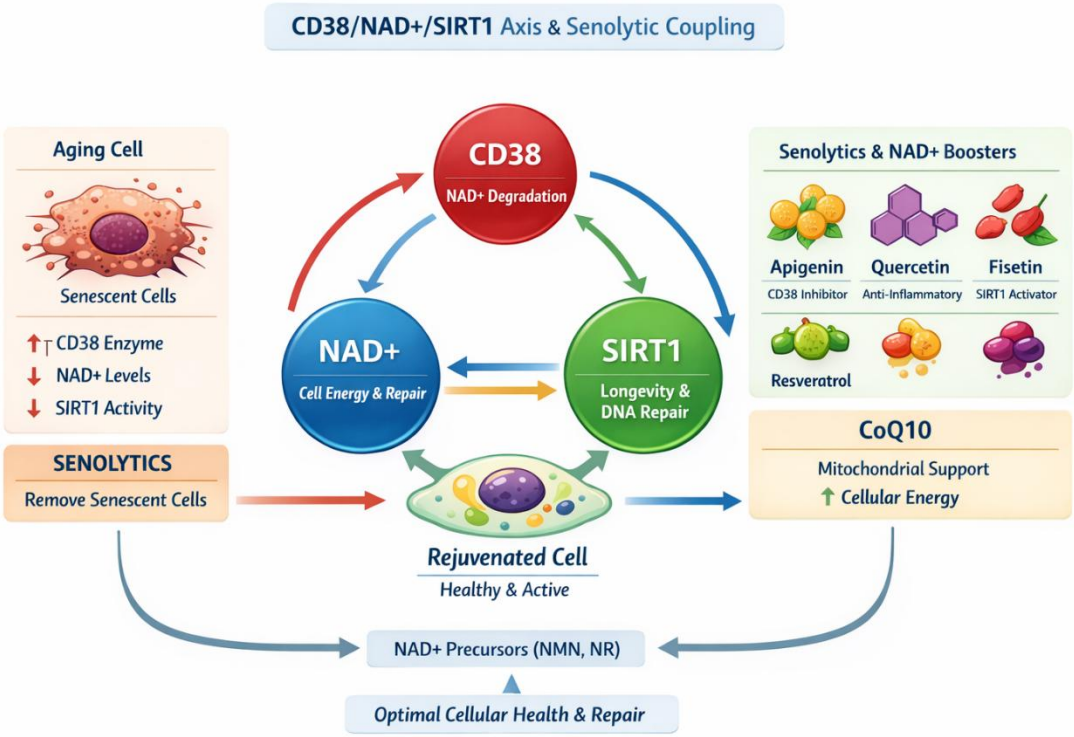

Fig. 2: **Senolytic Coupling with CD38/NAD+/SIRT1 Axis**

Beyond direct senolysis, the restoration of cellular homeostasis requires robust mitochondrial function, making **Coenzyme Q10 (CoQ10)** a critical adjunct in an integrated longevity protocol. **CoQ10 as a mitochondrial bioenergetic agent that works synergistically with senolytics.** While senolytics like Fisetin facilitate the clearance of dysfunctional cells, CoQ10 acts as a vital electron shunter within the mitochondrial respiratory chain, optimizing the production of adenosine triphosphate (ATP). By mitigating mitochondrial oxidative stress—a primary trigger for cellular senescence—CoQ10 prevents the transition of healthy cells into a senescent state. Furthermore, its synergy with the NAD+ pathway ensures that remaining healthy tissues have the bioenergetic capacity to replicate and repair the voids left by senolytic clearance, effectively bridging the gap between cell death and tissue rejuvenation

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6. Mechanism of Action (MOA)

The mechanism of action (MOA) for senolytics is centered on breaking down the "survival shields" that allow senescent cells to persist in the body. To understand how they work in simple terms, it is helpful to look at the unique state of a senescent cell and how senolytics intervene. When cells become damaged or stressed, they normally do one of two things: they either repair themselves or they undergo a process of programmed cell death called apoptosis. However, some cells enter a "zombie-like" state called senescence. In this state, they stop dividing but refuse to die. To survive this damaged state, these cells turn on specific "survival networks" known as Senescent Cell Anti-Apoptotic Pathways (SCAPs). These pathways act like a protective shield, making the cells resistant to the natural signals that would normally tell a damaged cell to eliminate itself. Senolytics are designed to selectively target and temporarily disable these specific survival shields. The process works in three simple steps:

1. Recognition: Senolytics are chosen because they interfere with the exact proteins (like BCL- or certain kinases) that senescent cells rely on to stay alive.
2. Disabling the Shield: Once the drug or natural compound enters the system, it "turns off" these anti-death signals.
3. Inducing Apoptosis: Without their survival shield, the senescent cells can no longer resist the pressure to die. They finally undergo apoptosis and are naturally cleared away by the body's immune system.

A key part of the senolytic MOA is selectivity. Because normal, healthy cells do not rely on these specific hyper-activated survival pathways, they are generally left unharmed by the senolytic treatment. This allows the body to "prune" away the harmful "zombie" cells that cause inflammation through their SASP (secretions) while leaving healthy tissue intact. By removing these "root cause" contributors to aging, senolytics help restore a healthier environment for the remaining young cells to thrive.

Natural senolytics work by disrupting the "defense shields" of senescent cells:

- Inhibition of SCAPs: They block pathways like PI3K/AKT and Bcl-xL that prevent SCs from undergoing apoptosis.
- SIRT1 Activation: Compounds like Quercetin activate Sirtuin-1, which regulates DNA repair and metabolic health.
- NAD+ Restoration: Many natural compounds work synergistically with NAD+ precursors (like NMN) to improve mitochondrial function and cellular energy.

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7. Multi-Systemic Therapeutic Effects.

The therapeutic potential of senolytics lies in their ability to target a fundamental biological "driver" of multiple diseases simultaneously. By eliminating senescent cells (SCs), these agents exert a broad range of beneficial effects across virtually every major organ system.

- **Anti-Aging and Tissue Rejuvenation**

The primary effect of senolytics is the extension of "healthspan." They clears SCs in the skin and joints, reducing frailty and promoting tissue regeneration .By pruning "zombie cells," these compounds allow tissues to regain their regenerative capacity. This manifests as reduced physical frailty, improved skin elasticity, and enhanced wound healing. In the musculoskeletal system, senolytics help maintain bone density and muscle mass, addressing the core physical declines associated with advancing age.

- **Anti-Cancer Effects**

Senescence is a double-edged sword in oncology. While it initially acts as a barrier to tumor growth, the long-term presence of SCs creates a "pro-tumor" environment. Senolytics help by Eliminates cells that have undergone oncogene-induced senescence, preventing them from turning into tumors. Furthermore, they can be used as an adjuvant therapy to clear senescent cells induced by chemotherapy, reducing the risk of cancer recurrence and treatment side effects.

- **Anti-Inflammatory and Antioxidant Actions**

Senolytics are powerful anti-inflammatory tools because they shut down the Senescence-Associated Secretory Phenotype (SASP)—the source of chronic "inflammaging." By removing the source of pro-inflammatory cytokines (like IL-6), they lower systemic inflammation. Additionally, they help restore the cellular redox balance, reducing oxidative stress and enhancing the body's natural antioxidant defenses. They neutralizes ROS and upregulates endogenous antioxidant enzymes

- **Anti-Obesity and Metabolic Health**

SC accumulation in adipose (fat) tissue is a major contributor to insulin resistance. Senolytics have been shown to improve metabolic function by reducing inflammation in fat cells, which enhances glucose uptake and combats obesity-related metabolic dysfunction. This makes them a promising strategy for managing Type 2 diabetes and non-alcoholic fatty liver disease. They improve insulin sensitivity and reduce adipose tissue inflammation.

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- **Anti-Cardiovascular Disease**

Senolytics reduce arterial stiffness and improve endothelial function. By clearing senescent vascular cells, they slow the progression of atherosclerosis and lower the risk of heart failure, essentially "rejuvenating" the circulatory system to a more youthful, flexible state.

Beyond these major categories, senolytics show promise in Neuroprotection (reducing brain inflammation linked to Alzheimer's) and Pulmonary Health (alleviating lung fibrosis). By targeting the root cause of systemic decline, senolytics represent a shift from treating individual symptoms to fostering holistic, multi-systemic health rejuvenation.

8. Advantages

Senotherapeutic's "hit-and-run" approach is revolutionary since these substances don't stay in body continuously. These are only taken occasionally to "clear out" the accumulation of zombie cells, unlike everyday treatments. This avoids the negative effects of long-term medication usage and keeps the liver and kidneys from being overworked. A single treatment can simultaneously boost heart, metabolism, and brain function by eliminating these "zombie cells"—a primary cause of aging. These are typically safe, inexpensive, and simple for the general population to utilize as part of a long-term health regimen because many of them are naturally occurring plant components (phytochemicals).

9. Limitations

Cellular heterogeneity, off-target toxicity in regenerative niches, and the low absorption of natural flavonoids currently limit the clinical usefulness of senotherapeutics despite their therapeutic potential. Additionally, the SASP's brief physiological benefit in wound healing raises the possibility that long-term senomorphic treatment could unintentionally compromise tissue-resident immune surveillance.

10. Conclusion and Future Perspective

Natural senolytics characterize a "food-as-medicine" approach in geroscience. These dietary phytochemicals offer a safer, more accessible alternate to synthetic drugs for health rejuvenation. The synergy between these plant-derived bioactives and NAD+ precursors offers a great strategy against "inflammation" and improved healthspan across age related diseases and disorders. Despite challenges like bioavailability, the development of functional "Nutri-senolytics" offers a proactive pathway to holistic health, shifting the medical paradigm from simply extending life to maintaining vibrant, disease-free vitality.

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By integrating these natural tools into clinical practice, we move closer to a future where aging is no longer an inevitable decline, but a manageable biological process.

Reference's

Sharma A, Chabloz S, Lapides RA, Roider E, Ewald CY. (2023). Potential Synergistic Supplementation of NAD+ Promoting Compounds as a Strategy for Increasing Healthspan. *Nutrients*. 14;15(2):445. doi:10.3390/nu15020445.

Imb M, Véghelyi Z, Maurer M, Kühnel H. Exploring (2024). Senolytic and Senomorphic Properties of Medicinal Plants for Anti-Aging Therapies. *International Journal of Molecular Sciences*. 25, 10419. https://doi.org/10.3390/ijms251910419

Dias, E & Poojary, D D (2024). Senolytics and Dietary Phytochemicals: Exploring Anti-Aging Strategies and Mechanisms in Aging and Disease. *International Journal for Multidisciplinary Research*. 6(3), 22349.

Rad A N, & Grillari J. (2024). Current senolytics: Mode of action, efficacy and limitations, and their future. *Mechanisms of Ageing and Development*, *217*, 111888. https://doi.org/10.1016/j.mad.2023.111888

Saliev, T & Singh P B. (2025). Targeting Senescence: A Review of Senolytics and Senomorphics in Anti-Aging Interventions. Biomolecules. 2025 Jun 13;15(6):860. doi: 10.3390/biom15060860.