

■ Cell Shedding Field Guide

Targeted Senolytic Clearing, Autophagy Activation & SASP Reduction

Executive Summary: Senescent cells are non-dividing 'zombie' cells that linger in tissue, secreting pro-inflammatory SASP cytokines that degrade surrounding cellular architecture. Periodic senolytic clearing restores youthful tissue dynamics.

Program Perspective & Biological Foundations

As cells age or undergo persistent DNA damage, they reach a biological crossroad: undergo apoptosis (programmed cell death) or enter a state of permanent arrest called senescence. Senescent cells refuse to die, lingering like 'zombies' in organ tissue.

While initial senescence prevents cancer, the long-term accumulation of senescent cells degrades healthy tissue. They secrete a toxic cocktail of inflammatory cytokines, chemokines, and tissue-destroying enzymes known as the Senescence-Associated Secretory Phenotype (SASP). Targeted senolytic clearing acts like seasonal pruning in an orchard, removing lingering zombie cells so stem cells can regenerate youthful tissue.

The Two Phases of Clearing

Phase I: Intermittent Senolytic Clearing ('Hit-and-Run')

Senescent cells depend on specialized Anti-Apoptotic Pathways (SCAPs) to survive. Administering targeted plant flavonoids (Fisetin, Quercetin) in 2-day monthly pulses temporarily disables SCAPs, inducing apoptosis specifically in senescent cells.

Phase II: Autophagy Activation & Lysosomal Recycling

Activating cellular self-cleaning (autophagy) between senolytic pulses ensures damaged organelle fragments and misfolded proteins are degraded and recycled into fresh amino acids.

The Three Exits & Transport Pathways

Toxins transformed by cellular metabolism must exit through three essential pathways. If any one exit stalls, the others cannot compensate fully:

1. Macrophage Phagocytosis — Clearing

Once a senescent cell undergoes senolytic-induced apoptosis, local tissue macrophages engulf and digest the cellular debris. Open transport pathways prevent inflammatory buildup.

2. Lysosomal Autophagy — Recycling

Mitochondria and proteins inside healthy cells undergo constant autophagy. Spermidine and intermittent fasting stimulate lysosomal enzymes to digest degraded sub-cellular structures.

3. SASP Suppression — Quenching

In between senolytic clearing pulses, botanical SASP dampeners (Curcumin, Boswellia, EGCG) suppress inflammatory cytokine release from remaining senescent cell populations.

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Targeted Supplement Protocol & Daily Schedule

Integrate targeted practitioner-grade formulations into your daily bio-rhythms:

Timing	Supplement & Form	Dose	Target Mechanism & Purpose
Pulse Days (Day 1 & 2)	Liposomal Fisetin	500–1,000 mg	High-potency senolytic flavonoid; selectively induces apoptosis in senescent cells
Pulse Days (Day 1 & 2)	Liposomal Quercetin	500 mg	Disrupts BCL-2/BCL-XL senescent cell anti-apoptotic survival pathways
Daily Maintenance	Spermidine (Trihydrochloride)	5–10 mg	Triggers lysosomal autophagy and mitophagy, recycling misfolded cellular proteins
Daily Maintenance	Curcumin Phytosome & Boswellia	500 mg	Potent SASP dampener; blocks NF-kB and downregulates pro-inflammatory cytokines
Post-Pulse Maintenance	Resveratrol & Pterostilbene	250 mg	Activates SIRT1 sirtuin pathways, stimulating progenitor stem cell tissue renewal

What to Expect Across the Protocol

Pulse (Days 1–2)

Senolytic flavonoids transiently disable zombie cell SCAP survival networks, triggering selective apoptosis in senescent cell populations.

Clearing (Days 3–7)

Macrophages clear apoptotic senescent cell debris. SASP dampeners suppress inflammatory cytokine spikes. Mild transient fatigue may occur.

Renewal (Days 8–30)

Tissue micro-environment rejuvenates. Progenitor stem cells expand, regenerating fresh, functional cell lines across targeted organs.

The Bigger Picture & Seasonal Integration

Cell Shedding is seasonal pruning. By clearing senescent cells in deliberate monthly or quarterly pulses, the body frees up vital energetic resources, reduces systemic inflammatory SASP burden, and allows fresh tissue renewal.

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