MERCOLA
Take Control of Your Health

Clearing Out 'Zombie' Cells: Exercise and Senolytics Restored Function in Aging Hearts

Analysis by Dr. Joseph Mercola | November 28, 2026

STORY AT-A-GLANCE

- A nine-month study in aging mice found that both regular aerobic exercise and a senolytic drug combination reduced the share of damaged, senescent cells in heart muscle and improved how well the heart pumped and relaxed
- Senescent cells, often called "zombie cells," stop dividing but refuse to die. They release inflammatory compounds that damage nearby tissue and push healthy neighboring cells into the same dysfunctional state
- Fisetin, a natural compound found in strawberries and apples, ranked as the most potent senolytic of 10 plant compounds tested in earlier research. A new 2026 study found it reduced senescent cells in aging blood vessels and restored their function in old mice
- Urolithin A complements senolytics by a different route. It helps your cells recycle damaged mitochondria and calms the inflammatory output of senescent cells, and it has several positive human trials
- My foundational strategy is regular exercise and a low linoleic acid diet, then urolithin A daily and a short, two-day fisetin pulse once a month to clear damaged cells

As you age, your body gradually accumulates cells that should have died but didn't. They're called senescent cells, and researchers increasingly view them as one of the central drivers of aging itself. Instead of quietly being removed, they linger in your tissues and release a steady stream of inflammatory compounds that damage everything around them.

Your heart is especially vulnerable. Heart muscle cells rarely divide, so damage accumulates over decades. As senescent cells build up, the heart muscle stiffens, relaxes less completely between beats and pumps less efficiently. Most people simply accept this as part of getting older.

A new study published in Aging Cell challenges that assumption.[1] It suggests that clearing senescent cells, either with exercise or with compounds called senolytics, can measurably improve function in an aging heart. I believe removing these damaged cells is one of the most important anti-aging strategies available today, and much of it is within your control.

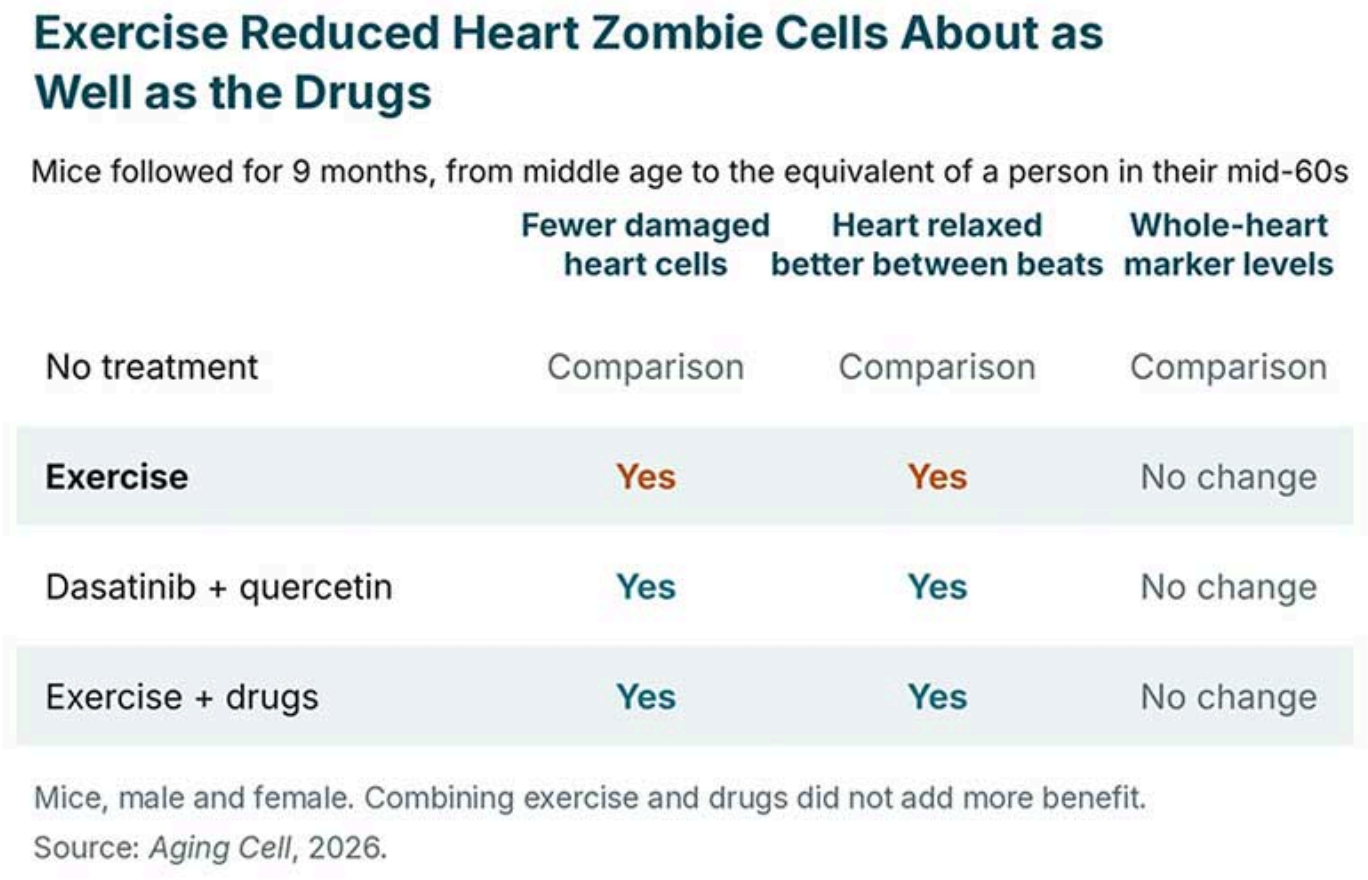
Exercise and Senolytics Both Improved Aging Hearts

Researchers at McMaster University and Brock University in Canada followed male and female mice from 12 months of age, roughly middle age, for nine months. That carried them to about 21 months, comparable to a person in their mid-60s. The mice were divided into four groups:

- No treatment
- Aerobic exercise
- The senolytic combination dasatinib plus quercetin
- Exercise and senolytics combined

- **Fewer damaged cells in the heart —** Exercise, senolytics and the combination all significantly lowered the proportion of heart muscle cells and surrounding support cells carrying markers of senescence and DNA damage. These markers included p16 and p21, two proteins that lock damaged cells into permanent growth arrest.
- **Better heart function —** All three treated groups showed improved heart muscle performance and better diastolic function. Diastolic function is the heart's ability to relax and fill with blood between beats, and it declines early with age.

- **Exercise held its own —** Exercise reduced senescence at the cellular level about as well as the drugs did. The researchers had predicted that combining the two would produce a dramatic additive effect. That didn't happen, which tells you how powerful exercise alone is.
- **What didn't change —** When the researchers measured senescence proteins and inflammatory signals across the whole heart rather than cell by cell, they found no significant change with any intervention. The benefits showed up in which individual cells remained damaged, not in total tissue levels of these markers.



Exercise Reduced Heart Zombie Cells About as Well as the Drugs

Mice followed for 9 months, from middle age to the equivalent of a person in their mid-60s

	Fewer damaged heart cells	Heart relaxed better between beats	Whole-heart marker levels
No treatment	Comparison	Comparison	Comparison
Exercise	**Yes**	**Yes**	No change
Dasatinib + quercetin	Yes	Yes	No change
Exercise + drugs	Yes	Yes	No change

Mice, male and female. Combining exercise and drugs did not add more benefit.
Source: *Aging Cell*, 2026.

Figure 1. In aging mice, exercise, dasatinib plus quercetin, and both together each reduced damaged heart cells and improved how the heart relaxed between beats.[2]

Why Zombie Cells Matter So Much

Senescence starts as a protective mechanism. When a cell's DNA is damaged beyond repair, shutting down permanently prevents it from turning cancerous. The problem comes when these cells accumulate faster than your immune system can clear them, which increasingly happens with age.

- **They poison their neighborhood —** Senescent cells release a mix of inflammatory and tissue-remodeling compounds known as the senescence-associated secretory phenotype. This chronic, low-grade inflammation stiffens tissues, promotes scarring and drives what researchers call "inflammaging."

- **They spread the damage —** Their secretions generate oxidative stress and can push healthy neighboring cells into senescence, creating a vicious cycle.

- **They resist dying —** Senescent cells survive by switching on internal defenses that block normal programmed cell death. That's their Achilles' heel.[3] Senolytics work by temporarily disabling these defenses, which lets the damaged cells finally die while healthy cells are left largely unharmed.

- **Their effects reach the mitochondria —** The oxidative stress senescent cells generate damages mitochondria, the energy factories in each cell, faster than your cells can recycle them. In your heart, which relies heavily on mitochondria for every contraction, energy output and pumping force fall.

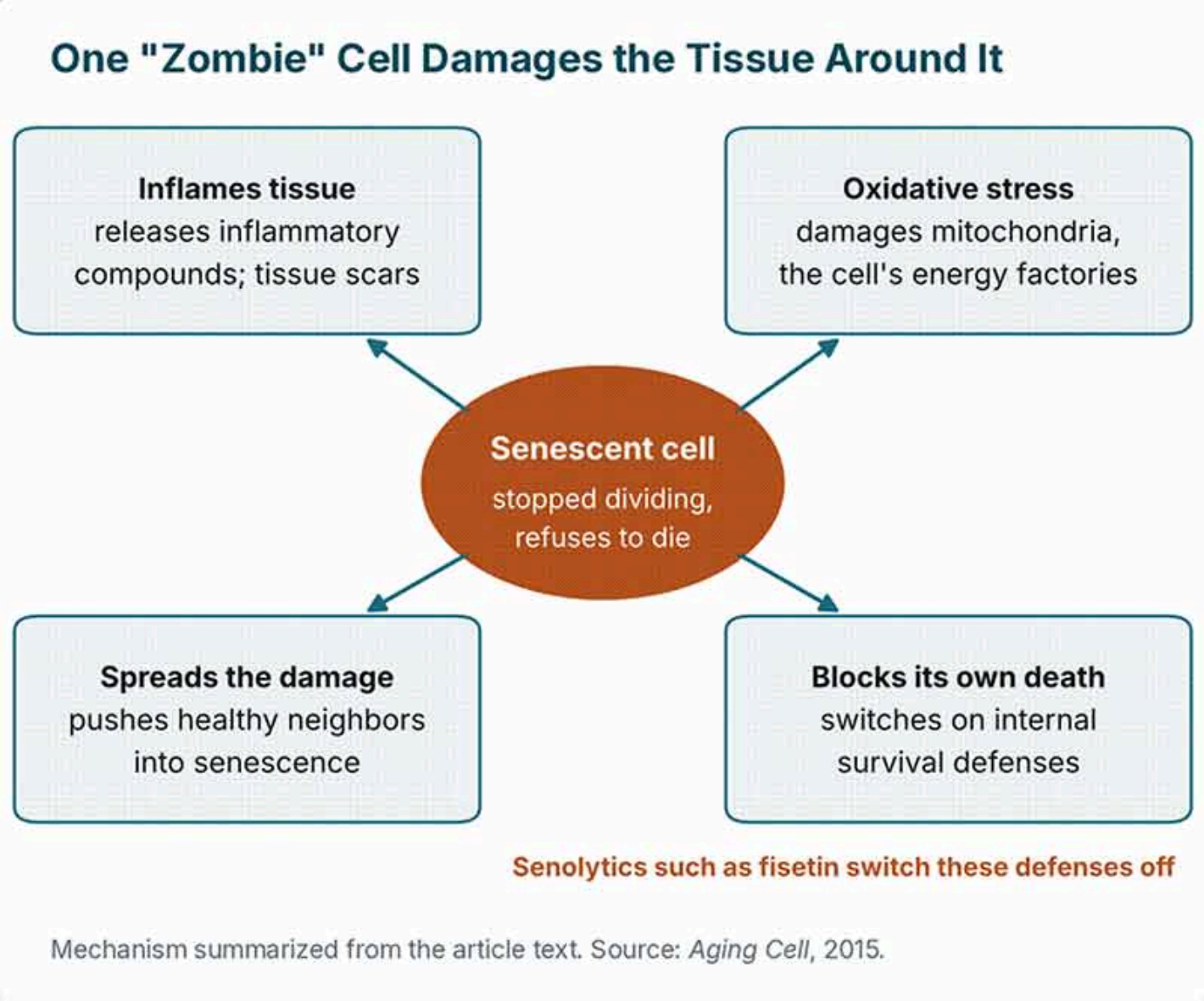


Figure 2. A senescent cell inflames and stiffens tissue, damages mitochondria, spreads senescence to its neighbors and blocks its own death, the defense senolytics switch off.[4]

The good news is that senolytics work in a "hit and run" fashion. They don't need to stay in your body. A short course triggers the death of senescent cells, which continues for days after the compound is gone. In the first human study to measure this directly, a single three-day course of dasatinib plus quercetin in nine people with diabetic kidney disease reduced senescent cells in fat and skin, measured 11 days later.[5] Because these cells reaccumulate slowly, a brief monthly pulse may be all that's needed.

The benefit also appears to depend on how many senescent cells you're carrying. In a 2024 Nature Medicine trial, postmenopausal women took dasatinib plus quercetin intermittently for 20 weeks.[6] Overall, it didn't reduce bone breakdown. But in an

exploratory analysis of the women with the highest senescent cell burden, it boosted bone formation and increased bone density at the wrist by 2.7%. The more damaged cells you have, the more you may stand to gain from clearing them.

Fisetin: The Senolytic from Your Fruit Bowl

The heart study used dasatinib, a prescription cancer drug, paired with quercetin. Dasatinib carries real side effects and isn't something to use on your own. Fortunately, there's a natural alternative with an impressive research record.

Fisetin, pronounced FISS-uh-tin, is a plant compound found in strawberries, apples, persimmons, and onions. When Mayo Clinic and Scripps researchers screened 10 plant compounds for senolytic activity, fisetin was the most potent.[7] Given to old mice late in life, it reduced senescence markers across multiple tissues and extended both median and maximum lifespan. Several studies published this year strengthen the case:

- **Restoring aging blood vessels —** In a 2026 Aging Cell study, old mice received fisetin at 100 milligrams per kilogram (mg/kg) daily for one week, then two weeks off, then another week.[8] Fisetin reduced senescent cells in the lining of their arteries. It sharply reduced a key inflammatory signal called CXCL12 and prevented the loss of nitric oxide, the molecule that keeps blood vessels flexible.

  When the researchers exposed healthy arteries to plasma from fisetin-treated old mice, the arteries dilated 14% better than with plasma from untreated old mice. Adding CXCL12 back eliminated the benefit, indicating that this inflammatory signal is one driver of age-related blood vessel dysfunction.

- **A human trial pairing fisetin with exercise —** In a 2026 randomized trial, 44 men with obesity took either 200 mg of fisetin daily or a placebo for 12 weeks, with or without combined resistance and aerobic training.[9] Fisetin alone lowered the inflammatory messengers IL-6 and TNF-alpha. The greatest improvements in blood sugar, insulin, and insulin resistance came from combining fisetin with training.

- **Calming brain inflammation —** In a mouse model of Alzheimer's disease, fisetin improved memory, reduced amyloid plaques, and calmed overactive brain immune cells.[10]

- **Beyond aging —** A senolytic cocktail including fisetin, added to standard TB drugs, improved survival in mice with tuberculosis,[11] and topical fisetin improved wound healing in diabetic mice and reduced some senescence markers and inflammation in the skin.[12]

The Absorption Problem

Fisetin has one significant weakness: your body absorbs very little of it. In a human study, a 1,000-mg dose of plain fisetin produced a peak blood level of only about 10 nanograms per milliliter. An enhanced formulation delivering just 192 mg produced levels about 24 times higher.[13]

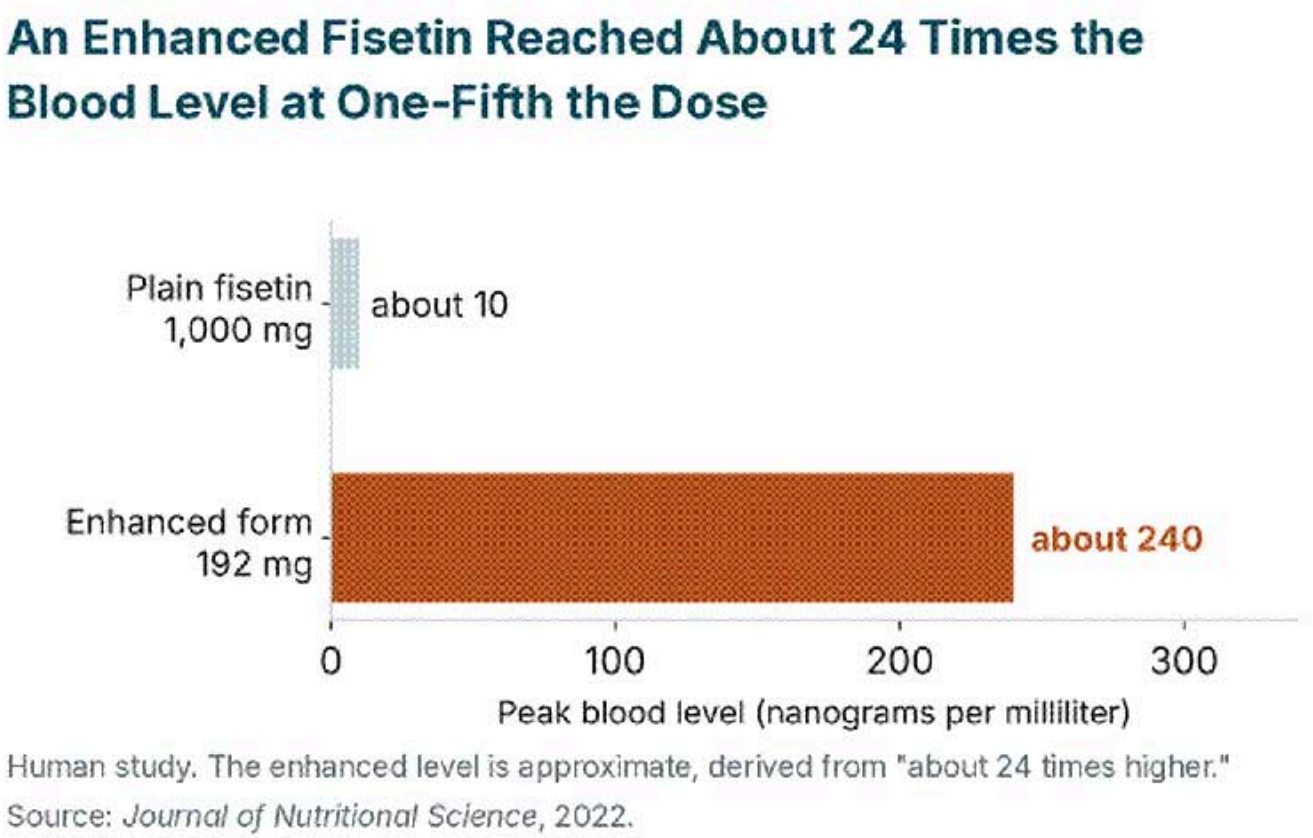


Figure 3. A 1,000 mg dose of plain fisetin reached a peak blood level of about 10 nanograms per milliliter; an enhanced 192 mg formulation reached about 24 times higher.[14]

Urolithin A: Cleaning Up Damaged Mitochondria

If senolytics remove damaged cells, urolithin A may help prevent cells from becoming damaged in the first place. It's produced naturally when gut bacteria break down compounds in pomegranates, berries and walnuts. Many people's gut bacteria make little or none, so supplementing provides it directly.

Urolithin A activates mitophagy, the process your cells use to identify and recycle worn-out mitochondria. Since mitochondrial dysfunction is one of the triggers that pushes cells into senescence, this addresses the problem upstream. Its human evidence is the strongest of any compound discussed here:

- **Stronger muscles —** In middle-aged adults, four months of urolithin A improved muscle strength by about 12%, along with endurance and markers of mitochondrial health.[15]

- **Better endurance in older adults —** In adults aged 65 to 90, 1,000 mg daily improved muscle endurance in the hands and legs.[16]

- **A younger immune system —** In a 2025 Nature Aging trial in 50 healthy middle-aged adults, four weeks of urolithin A at 1,000 mg daily expanded the population of fresh, naive immune T cells and improved their mitochondria.[17] A stronger immune system may be better at clearing senescent cells on its own.

- **Calming zombie cells —** Laboratory research shows urolithin A also quiets the inflammatory output of existing senescent cells, partly by preventing DNA fragments leaking from damaged nuclei and mitochondria from triggering inflammation.[18]

How to Clear Damaged Cells and Protect Your Body

These four strategies work together. Exercise and a low linoleic acid diet reduce the damage that creates senescent cells, urolithin A keeps your mitochondria healthy, and fisetin periodically clears the damaged cells that accumulate anyway. I'm also working

on several other approaches that I believe may prove even more powerful, but these are the foundation.

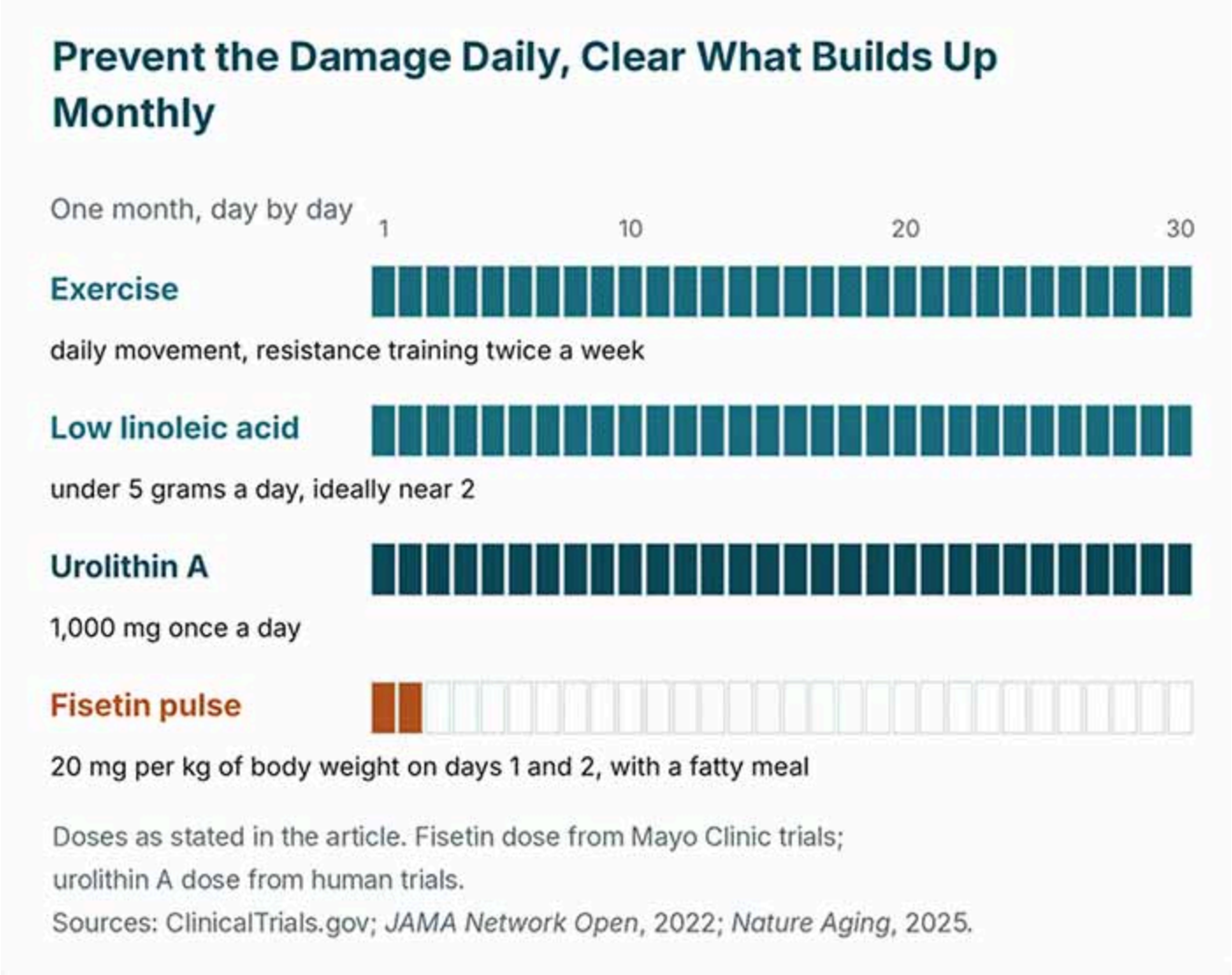


Figure 4. Exercise, a low linoleic acid diet and urolithin A work every day of the month; fisetin is taken on just two days.[19,20,21]

- **Exercise regularly —** This study shows exercise reduced senescent cells in the hearts of aging mice about as effectively as the drugs did. Aim for daily movement, such as a brisk walk, along with resistance training two times a week. The fisetin trial found the largest metabolic improvements when supplementation was combined with training.

- **Lower your linoleic acid intake —** This is just as foundational as exercise. Linoleic acid, the omega-6 fat concentrated in seed oils like soybean, corn, canola and sunflower oil, accumulates in your cell membranes, including your mitochondria.

Because it is highly prone to oxidation, it fuels the kind of oxidative damage that harms mitochondria and DNA, the very damage that pushes cells into senescence. Replace seed oils with stable fats such as grass fed butter, ghee, and tallow. Keep your linoleic acid intake below 5 grams a day, and ideally closer to 2 grams.

- **Consider urolithin A daily —** Most of the human trials used 1,000 mg once a day, including the studies in older adults and on immune aging. Because mitophagy needs to happen continually, this is a daily supplement rather than a periodic one.

- **Use fisetin as a monthly pulse —** The dose used in Mayo Clinic clinical trials is 20 mg per kilogram of body weight daily for two consecutive days.[22] For someone weighing 176 pounds (80 kg), that's about 1,600 mg a day. One of the trials repeated it the following month. Taking it with a fatty meal may improve absorption.

  During those two days, cut your caffeine intake roughly in half, as the Mayo protocol advises, because fisetin slows how your body clears caffeine. Daily low doses of fisetin appear to act more as an anti-inflammatory than a senolytic, so keep the high-dose pulse brief.

  If you take prescription medications, particularly blood thinners, check with your doctor before using fisetin, as it can affect how your liver processes some drugs. Avoid it during pregnancy.

These findings are from laboratory or animal research and may not directly apply to human health.

This article is for informational purposes only and does not constitute medical advice. Consult a qualified healthcare provider before making changes to your health regimen.

Frequently Asked Questions

Q: What are senescent cells?

A: Senescent cells are damaged cells that have permanently stopped dividing but resist dying. They release inflammatory compounds that damage surrounding tissue and push healthy neighboring cells into senescence. They accumulate with age and are increasingly viewed as a major driver of aging and age-related disease.

Q: What did the new heart study find?

A: In aging mice followed for nine months, aerobic exercise, the senolytic combination dasatinib plus quercetin, and both together reduced the proportion of senescent cells in heart muscle and improved heart function, including the heart's ability to relax between beats. Exercise alone performed about as well as the drugs at the cellular level.

Q: How do senolytics work?

A: Senescent cells survive by switching on internal defenses that block normal cell death. Senolytics temporarily disable those defenses, allowing the damaged cells to die. Because the effect continues after the compound leaves your body, a short course once a month may be enough.

Q: Why choose fisetin?

A: Fisetin is a natural plant compound that ranked as the most potent senolytic of 10 tested in laboratory research. It extended lifespan in old mice, and new 2026 research shows it reduces senescent cells in aging blood vessels and restores their function.6 It has a favorable safety profile, unlike prescription senolytics.

Q: How should I take fisetin and urolithin A?

A: For fisetin, the clinical trial dose is 20 mg per kilogram of body weight daily for two consecutive days, repeated monthly. That's about 1,600 mg a day for someone who weighs 176 pounds. Taking it with a fatty meal may help, since it's poorly absorbed on its own. For urolithin A, most human trials used 1,000 mg once a day.

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