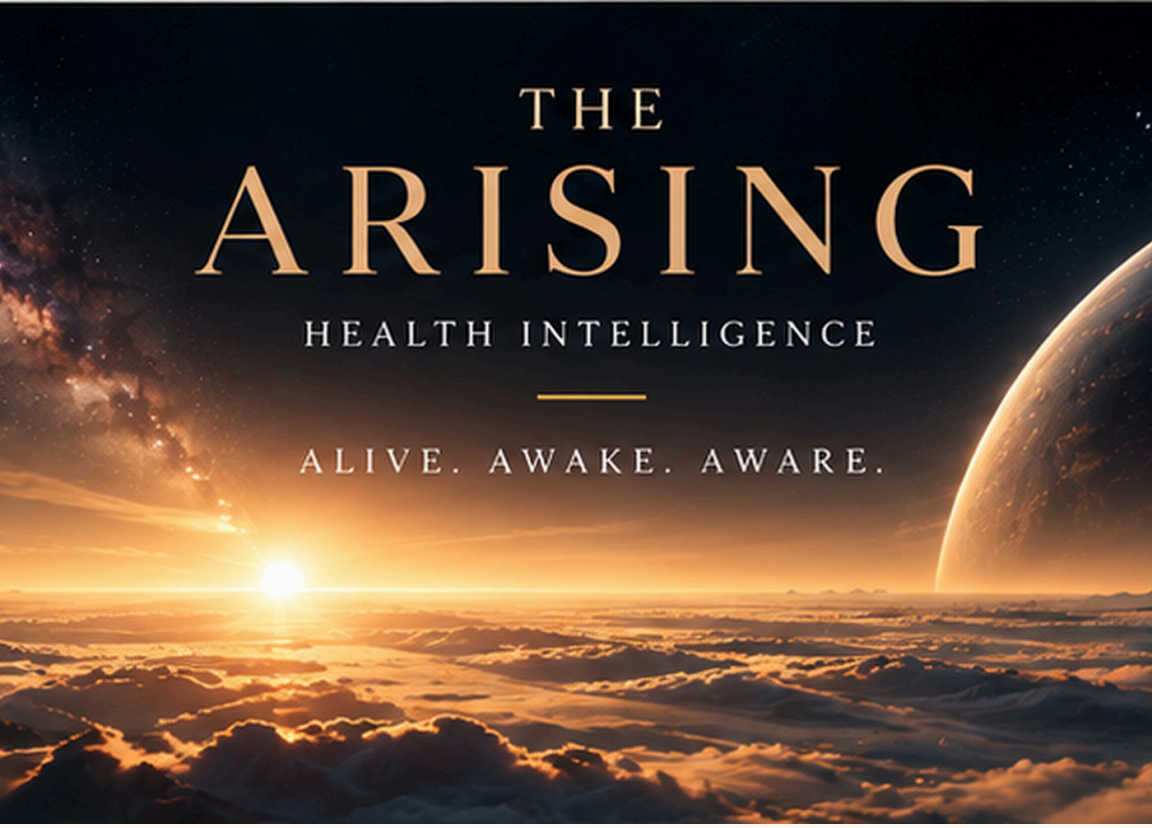


23 / BIOLOGY AND REPAIR

The Cells That Stay Too Long

Senescence, senolytic research, and how an early human signal should be read.

JOHN GRIFFIN, MD

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One name, several biological states

Senescence is a useful research concept, but it should not turn every aging cell into the same target. The relevant cell type, tissue, stage of disease, and method of identification all matter when deciding what an experiment can establish.

The language of “zombie cells” makes the field easy to remember. It can also conceal the difficult work: showing which cells should be changed or removed, what the intervention actually reaches, and whether the tissue functions better afterward.

An early human study

A 2019 open-label pilot studied dasatinib plus quercetin in 14 people with idiopathic pulmonary fibrosis. Its primary purpose was feasibility. Some physical-function measures improved, while pulmonary function and several other measures did not. One serious adverse event was reported. There was no randomized control group. [1]

The study justified further investigation. It could not establish that treatment caused the functional changes, that it slowed pulmonary fibrosis, or that it extends life in generally healthy adults. A drug combination also retains its risks when discussed in a longevity setting.

Removing a cell is only one possible route

Research on chaperone-mediated autophagy suggests that senescent-cell persistence can involve impaired clearance by macrophages. In the reviewed cell and mouse experiments, changing a maintenance pathway affected that relationship. [2]

This broadens the research question. A tissue may be affected by the production of senescent cells, their properties, their removal, or communication with surrounding cells. Those approaches need distinct tests.

What would change confidence

A convincing clinical result would need an appropriate comparator, evidence that the intended target was affected, meaningful outcomes, and adequate follow-up for harms. A favorable molecular signature alone would leave much unresolved.

For now, senolytics belong in an evidence conversation with careful attention to the study population and trial stage. The possibility is substantial enough to investigate. Its importance is a reason to demand a clearer answer, not a reason to treat an early experiment as a personal protocol.

SOURCES & READING SCOPE

Keep the claim beside its evidence

1. Jamie N Justice, Anoop M Nambiar, Tamar Tchkonia, et al. Senolytics in idiopathic pulmonary fibrosis: Results from a first-in-human, open-label, pilot study. EBioMedicine. 2019 Feb;40:554-563. doi:10.1016/j.ebiom.2018.12.052. PMID:30616998. Open source →

2. Rebecca Sereda, Kristen Lindenau, Antonio Diaz, et al. Decline of chaperone-mediated autophagy in aging impairs macrophage clearance of senescent cells. Nature aging. 2026 Oct 05:10.1038/s43587-026-01240-w. doi:10.1038/s43587-026-01240-w. PMID:42834177. Open source →

Selected primary publication records and abstracts checked 25–26 October 2026. This is a focused reading companion, not a systematic review, a treatment protocol, or a complete appraisal of methods, supplements and later evidence.

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