



Cognitive Vitality Reports® are reports written by neuroscientists at the Alzheimer's Drug Discovery Foundation (ADDF). These scientific reports include analysis of drugs, drugs-in-development, drug targets, supplements, nutraceuticals, food/drink, non-pharmacologic interventions, and risk factors. Neuroscientists evaluate the potential benefit (or harm) for brain health, as well as for age-related health concerns that can affect brain health (e.g., cardiovascular diseases, cancers, diabetes/metabolic syndrome). In addition, these reports include evaluation of safety data, from clinical trials if available, and from preclinical models.

Senolytics

Evidence Summary

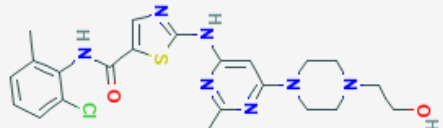
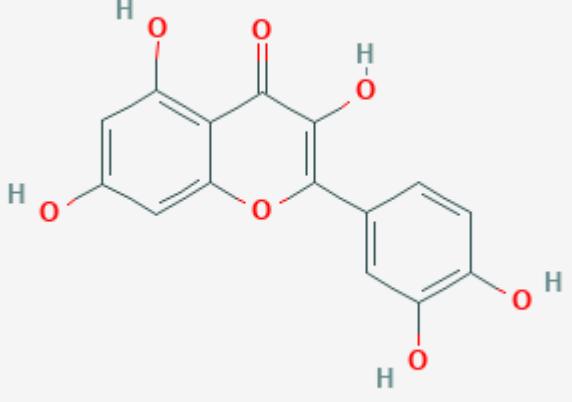
Evidence suggests that senolytic drugs may promote healthspan. Multiple clinical trials are underway.

Neuroprotective Benefit: Some evidence suggests an increase in senescent cells in Alzheimer's disease, though the identification of which cells are senescent and whether senolytic treatment would be beneficial are still uncertain.

Aging and related health concerns: Preclinical evidence suggests that clearance of senescent cells may promote lifespan and certain aspects of healthspan – clinical trials are underway.

Safety: Most senolytics are cancer drugs with potentially serious side effects, though the effects of a 'hit and run' approach are unknown.

Two common senolytics – Quercetin and Dasatinib

<p>Availability: Quercetin is available as a supplement, dasatinib as a prescription drug; other senolytic agents are available as supplements, prescription drugs, or experimental compounds</p>	<p>Dose: Dose used in one clinical trial: dasatinib (100mg/day), quercetin (1250mg/day) three consecutive days in a week for three weeks</p>	<p>Chemical formula: Dasatinib: $C_{22}H_{26}ClN_7O_2S$ MW: 488.006g/mol Source: Pubchem</p> 
<p>Half life: 1.3-5 hours (dasatinib) 3.5 hours (quercetin)</p>	<p>BBB: Penetrant in animals (D+Q)</p>	<p>Quercetin: $C_{15}H_{10}O_7$ MW: 302.238g/mol Source: Pubchem</p> 
<p>Clinical trials: One clinical trial completed using D+Q in idiopathic pulmonary fibrosis</p>	<p>Observational studies: None</p>	



What is it?

In response to certain stressors, including genotoxic stress (i.e. irradiation, chemotherapeutic drugs, etc.), oncogenic stress (activation of oncogenes), and telomere erosion (i.e. reaching the Hayflick limit), cells may permanently stop dividing and undergo senescence. This phenomenon may have evolved as a safeguard against cancer by preventing unrestrained proliferation of damaged cells. In normal circumstances the immune system clears these cells from the body. However, as we age, these cells may accumulate in certain tissues – such as fat, muscle, and bone marrow – where they secrete pro-inflammatory cytokines, chemokines, and extracellular matrix proteins ([Childs et al, 2015](#)). This is called the senescence-associated secretory phenotype – or SASP ([Zhu et al, 2015](#)). SASP can affect biological processes in surrounding tissue including cell proliferation, angiogenesis, and inflammation. It can also lead surrounding cells to become senescent. Therefore, reducing SASP by eliminating senescent cells might promote health in aged tissue.

Senescent cells can be identified in several ways including expression of senescence-associated β -galactosidase (SA β -gal), p16^{INK4a}, *Cdkn2a*, p21, markers of DNA damage, nuclear loss of laminin B1 or HMGB1 – though markers are not always associated with senescence and not all senescent cells express the same markers. It is recommended to confirm senescence with multiple markers ([Baker and Petersen, 2018](#)).

Current senolytic treatments

Genetic mouse models:

INK-ATTAC: expresses an inducible caspase 8 under the p16^{INK4a} promoter to induce apoptosis of senescent cells with administration of AP20187

P16-3MR: express a truncated herpes simplex virus thymidine kinase under the p16^{INK4a} promoter to induce apoptosis on administration of ganciclovir (GCV)

Small molecules include: dasatinib, quercetin, fisetin, luteolin, enzastaurin, navitoclax, A1331852, A1155463, piperlongumine, FOXO4-related peptide, nutlin-3a, geldanamycin, tanexpimycin, alvespimycin

A gene therapy and antibody approach are also being developed.



Neuroprotective Benefit: Some evidence suggests an increase in senescent cells in Alzheimer's disease, though the identification of which cells are senescent and whether senolytic treatment would be beneficial are still uncertain.

Types of evidence:

- 3 post-mortem studies of Alzheimer's brain tissue
- 3 preclinical senolytic studies

Human Evidence:

Post-mortem tissue – Alzheimer's

The first report of senescent cells in the brain found that nearly 50% of the astrocytes in the frontal cortex also expressed the senescent marker p16^{INK4a} ([Bhat et al, 2012](#)).

[Zhang et al \(2019\)](#) reported an increase in senescent oligodendrocyte progenitor cells (OPCs) surrounding amyloid plaques in patients with Alzheimer's disease but not MCI or normal controls. They found no increase in senescence in plaque-associated astrocytes or microglia.

Using data from a publicly available database, [Musi et al \(2018\)](#) reported that neurons containing neurofibrillary tangles had changes in gene expression indicative of cell senescence (e.g. upregulation of cell survival pathways, inflammation, cell cycle progression; downregulation of cell death pathways) compared to adjacent neurons. They also reported a 57% increase in p16 mRNA in brain tissue from patients with progressive supranuclear palsy (PSP), an age-related tauopathy.

Markers of SASP (i.e. the inflammatory marker IL-6) are also elevated in Alzheimer's and Parkinson's disease tissue and CSF – though this may not be specific for cellular senescence ([Tan et al, 2014](#)).

Parkinson's

[Chinta et al \(2018\)](#) reported an increase in senescent markers ($p16^{INK4a}$, IL-6, IL-1 α , IL-8, MMP3) in the substantia nigra of patients with sporadic Parkinson's disease which was specific for astrocytes (Lamin B1 staining in GFAP+ cells).

Mechanism of action for benefit identified from laboratory and preclinical studies:

Evidence for the presence of senescent cells

In an Alzheimer's mouse model, [Zhang et al \(2019\)](#) found an increase in senescent cells around amyloid plaques but not in plaque free areas. The senescent cells were OPCs, and not astrocytes, microglia, or



oligodendrocytes. In cell culture studies they found that A β could increase senescence in OPCs and that Olig2, an OPC transcription factor, loses its nuclear localization. They found little myelin in areas associated with plaques and suggest that OPCs undergo replicative senescence in the Alzheimer brain.

In a tau mouse model, brain tissue developed a senescence-associated profile (e.g. increased DNA damage, NF κ B activation, and upregulation of SASP), there was a reduction of mitochondrial respiratory capacity, and an increase in the expression of proteins associated with senescent cells (e.g. p65, Cdkn2a). This was reported to be specific for neurofibrillary tangles, as an Alzheimer's mouse model did not have an increase in a marker of senescence (*Cdkn2a*) at a stage after plaque accumulation but before neurofibrillary tangle accumulation ([Musi et al, 2018](#)).

In contrast, [Bussian et al \(2018\)](#) found an increase in *Cdkn2a* in a tau mouse model that began before the onset of neurofibrillary tangles. They found that senescent cells (SA- β -Gal) were microglia and astrocytes, but not neurons or oligodendrocytes, and found double the number of senescent cells in the hippocampus and cortex of tau mice compared to controls.

Senolytic treatment – Alzheimer's

Dasatinib (12mg/kg) and quercetin (50mg/kg) entered the brain after acute treatment. Nine-day treatment in 5-month-old Alzheimer's mice reduced the number of plaque-associated senescent cells and plaque-associated OPCs but had no effect on plaque load or astrocyte/microglia number. D+Q altered the 'activation morphology' of microglia, but not astrocytes, and reduced levels of IL-6. Chronic treatment with D+Q (once/week for 11 weeks) in 3-month-old Alzheimer's mice improved cognition, reduced A β levels, reduced plaque load, reduced plaque-associated OPCs (but not astrocytes or microglia), and reduced inflammation cytokines (TNF α , IL-1 β , and IFN γ) ([Zhang et al, 2019](#)).

In an aged tau animal model, biweekly D+Q over 3 months reduced the number of neurofibrillary-containing neurons by 35%, decreased ventricular volume by 28%, and reduced cortical atrophy. Additionally, D+Q increased the expression of a marker of synapses and neurons (though it is not clear whether it increased the number of neurons or just the expression of the marker) ([Musi et al, 2018](#)).

Twice-weekly administration of AP20187 to a tau mouse model with the *ATTAC* transgene (AP20187 eliminates senescent cells in *ATTAC* mice) from young adulthood prevented the increase in the expression of senescent gene expression with age. Additionally, AP20187 treatment reduced the number of inflammatory microglia and astrocytes (*GFAP*, *S100 β* , *Cd11b*), the levels of phosphorylated tau, and the number of neurofibrillary tangles. AP20187 treatment also prevented whole brain atrophy



and hippocampal neuronal loss and improved cognitive function. Early treatment with navitoclax (50mg/kg for five days followed by 16 days rest) similarly prevented senescent cell accumulation and tau pathology ([Bussian et al, 2018](#)).

Parkinson's

In a Parkinson's model (systemic paraquat treatment), there was an increase in cellular senescence in the striatum ($p16^{INK4a}$, $IL-6$) that was specific for astrocytes. Elimination of senescent cells (p16-3MR-GCV model), reduced the number of senescent cells, reduced neuronal loss, increased neurogenesis (in the subventricular zone), and improved behavior ([Chinta et al, 2018](#)).

ApoE4 Interactions: None reported

Aging and health-related concerns: Preclinical evidence suggests that clearance of senescent cells may promote lifespan and certain aspects of healthspan – clinical trials are underway.

Types of evidence:

- One clinical study of D+Q in idiopathic pulmonary fibrosis
- Multiple preclinical studies in age-related conditions

Human Evidence:

The dasatinib (D) + quercetin (Q) combination is the only senolytic to complete a clinical trial with reported results. In an open-label study, patients with idiopathic pulmonary fibrosis (n=14) were treated with D (100mg/day) + Q (1250mg/day) three days per week for three weeks. Some measures of physical function improved (6-minute walk test, 4-minute gait speed, timed chair-stands) though measures of lung function did not improve nor did a frailty score (though these were not expected to change due to the short time frame). Markers of SASP in the plasma did not substantially change, though the authors mention they were not that elevated to begin with. Notably, no patients dropped out due to side effects ([Justice et al, 2019](#)).

Mechanism of action for benefit identified from laboratory and preclinical studies:

Experiments with AP20187:

Healthspan/Lifespan Benefit: AP20187 treatment every three days in a 3-week old mouse model of accelerated aging (a cross between $INK-ATTAC$ and $BurbR1^{H/H}$) decreased senescent cells in fat, muscle,



and eye tissue and delayed the incidence of age-related fat loss, sarcopenia, and cataracts ([Baker et al, 2011](#)). When AP20187 was administered for 5 months to older mice of the same strain – at a time when age-related phenotypes were already present – markers of senescence were reduced, and age-related muscle and fat loss was attenuated. However, cataracts, which had already formed by 5 months, remained.

This mouse model of accelerated aging suffered from cardiac problems, and the inducible transgene is not expressed in cardiac tissue in these mice. Therefore, AP20187 treatment resulted in no increase in lifespan. [Baker et al \(2011\)](#) speculated the mice died from cardiac disease. Therefore, [Baker et al \(2016\)](#) treated regular 12-month-old *INK-ATTAC* mice with AP20187 until 18 months. Treatment reduced age-related fat loss, reduced kidney damage, slowed age-related decline in exploratory behavior and increased resistance to cardiac stress. However, it did not improve motor/muscle performance, measures of metabolic parameters, and other measures of cardiac performance compared to untreated mice. In lifespan studies, [Baker et al \(2016\)](#) reported that treatment with AP20187 increased median lifespan by 17%-35%, depending on the strain and sex of the mouse and increased maximum lifespan in a subset of mice.

p16-3MR

Atherosclerosis Benefit: In a mouse model of atherosclerosis (*Ldlr*^{-/-}; high fat diet – HFD), senescent cells (endothelium-like, vascular smooth muscle cell-like, and foamy macrophage-like) accumulated within the plaque area but not in the surrounding vasculature. Short-term treatment with GVC cleared the plaque-associated senescent cells. Senescent cell clearance starting at the beginning of the HFD reduced plaque burden (~60%), plaque size, and prevented the destruction of the aortic elastic fibers beneath the neointima in the brachiocephalic artery (a site of rapid plaque development). Senescent cells in the aorta were also reduced. There were no changes in circulating immune cells or atherogenic lipids suggesting the mechanism was specific for the reduction of senescent cells. These results were confirmed in two transgenic systems (*INK-ATTAC* and *INK-Nitroreductase*) and using navitoclax (100mg/kg). Clearance of senescent cells prevented plaque development when cleared early and prevented plaque progression and promoted plaque stability when cleared later after plaque development ([Childs et al, 2016](#)).

The experiments with AP20187 and GCV provided proof-of-concept data that clearance of senescent cells may prevent some age-related phenotypes of certain tissues. Efforts are underway to discover senolytic drugs that can eventually be used in humans. Screens for these drugs are conducted in particular cell types (e.g. senescent pre-adipocytes and senescent endothelial cells) and certain drugs

show preference for clearing senescent cells in particular tissues. Generation of the first class of senolytics was hypothesis-based – e.g. targeting pro-survival pathways that are present in cancer cells.

UBX0101 – Unity Biotechnology (mechanism unreported)

Osteoarthritis Benefit: Clearance of senescent cells in an osteoarthritis injury model using the *p16-3MR* model and intra-articular injection of another senolytic, UBX0101, reduced local inflammation, reduced pain, and improved functional outcomes. The half-life of UBX0101 was short (1.5 hours) and elimination of senescent cells was achieved after 5 injections given every two days. However, 82 days after the final injection, joint pain increased, suggesting a return of senescent cells into the intra-articular space. UBX0101 was also effective when injected 42 days after the injury. Clearance of senescent cells (using the *INK-ATTAC* mice) also reduced age-related osteoarthritis in older mice ([Jeon et al, 2017](#)).

UBX0101 also cleared 20% of senescent cells in human osteoarthritis cartilage explants ([Jeon et al, 2017](#)). In a proof-of-concept study, [Jeon et al \(2019\)](#) found that treatment of a mouse model of osteoarthritis with UBX0101 altered the miRNA profile of extracellular vesicles in the synovial fluid.

Dasatinib and Quercetin:

Dasatinib (D) and quercetin (Q) were discovered in an *in vitro* screen for drugs that promote apoptosis in senescent human pre-adipocytes and human umbilical vein endothelial cells (HUVECs). Dasatinib cleared senescent pre-adipocytes better while quercetin cleared senescent HUVECs better.

Healthspan/Lifespan Benefit: A single treatment of D+Q (D: 5mg/kg; Q: 50mg/kg) reduced senescent cells in fat, liver, and irradiated muscle tissue (irradiation is used to promote senescence) of 24-month-old mice within 5 days. Treatment of D+Q also modestly improved some measures of cardiac function (but not others) and, in a mouse model of accelerated aging, improved some measures of healthspan (especially movement dysfunctions) ([Zhu et al, 2015](#)).

IP injection of senescent preadipocytes in middle-age and old mice reduced physical function after one month (speed, hanging endurance, and grip strength). In old mice, IP injection of senescent preadipocytes accelerated time to death – especially due to non-cancer causes. D+Q reduced senescent cell abundance and SASP in human adipose tissue explants. 3-day treatment with D (5mg/kg) +Q (50mg/kg) prevented and rescued the physical deficits that occurred after IP injection of senescent preadipocytes. Finally, biweekly treatment with D+Q in old (20 month) mice improved physical function after 4 months, and, in very old mice (24 months) increased median lifespan by 36% ([Xu et al, 2018](#)).



Atherosclerosis Benefit: D+Q reduced senescent cell markers in the medial layer of the aorta in aged and hypercholesterolemic mice, but not in intimal atherosclerotic plaques. In hypercholesterolemic mice, plaque calcification, but not plaque size or lipid content, were reported to be reduced after 2 months of D+Q treatment ([Roose et al, 2016](#)).

Diabetes Benefit: In a mouse model of diabetes (db/db and high fat diet), intermittent D (5mg/kg) + Q (50mg/kg) treatment (5 consecutive days every month) reduced the number of senescent cells, improved glucose homeostasis, improved insulin sensitivity, and improved renal function. The improvements in insulin sensitivity were thought to be because new adipose cells derived from the remaining preadipocytes are more insulin sensitive. Similar results were seen with other methods of senolytic treatment (*INK-ATTAC, p16-3MR*) ([Palmer et al, 2019](#)).

Bone loss Benefit: D+Q also eliminated senescent cells in the bone and adipose tissue of old mice, and treatment for four months beginning at old age prevented some of the age-related bone loss ([Farr et al, 2017](#)).

FOXO4-p53 peptide

Healthspan Benefit: [Baar et al \(2017\)](#) found that FOXO4 is increased in senescent cells, and that inhibiting the interaction of FOXO4 with p53 induced the apoptosis of senescent cells. The FOXO4-p53 peptide also improved physical activity in a mouse model of accelerated aging and improved fur density and renal function in a model of accelerated aging and in naturally aged mice.

Piperlongumine

Piperlongumine induced apoptosis of senescent human fibroblasts *in vitro* ([Wang et al, 2016](#)). Further studies suggested it induced apoptosis by inhibiting oxidative resistance 1 (OXR1) causing senescent cells to be more sensitive to oxidative stress ([Zhang et al, 2018](#)).

Fisetin

Healthspan/Lifespan Benefit: In a screen of potential senolytics, fisetin was reported to be senolytic for mouse and human fibroblasts. Two two-week treatments with fisetin in the diet (equivalent to about 60mg/kg per day) reduced the number of senescent cells in a model of accelerated aging. Starting fisetin in the diet late in life (at 85 weeks – equivalent to about 75 years in humans) increased median lifespan (from ~27 to ~30 months) and improved healthspan (e.g. liver and immune function). Fisetin also reduced senescent cell in human adipose tissue explants ([Yousefzadeh et al, 2018](#)).



Navitoclax:

Healthspan/Cardiovascular Benefit: Another senolytic drug, navitoclax (also known as ABT-263), was discovered in an *in vitro* screen of senescent HUVECs, human lung fibroblasts, and murine embryonic fibroblasts. However, it did not reduce the viability of senescent human preadipocytes *in vitro* (unlike dasatinib and quercetin; [Zhu et al, 2016](#)). [Chang et al \(2016\)](#) reported that navitoclax reduced senescent cells in the lungs, bone marrow, and muscles of aged mice and young irradiated mice. Navitoclax also reduced the number of senescent cells in the heart of old mice, and treatment of navitoclax (50 mg/kg per day – two 7-day treatments) prior to experimental myocardial infarction reduced mortality (from 40% survival to >90%) and improved heart function ([Walaszczyk et al, 2019](#)).

EF24

EF24, a curcumin analog, was shown to be senolytic *in vitro* in human fibroblasts, HUVEC, HREC (human renal endothelial cells), and human preadipocytes. It had synergistic senolytic effects with navitoclax in fibroblasts ([Li et al, 2019](#)).

Azithromycin and roxithromycin

Azithromycin and roxithromycin were reported to be senolytic for human lung fibroblasts *in vitro* ([Ozsvari et al, 2018](#)).

ABT-737:

[Yosef et al \(2015\)](#) reported that 2 day or 4-day treatment of ABT-737, another potential senolytic drug, reduced senescent cells in lungs and increased the proliferation of hair stem cells in irradiated mice. It also removed genetically-induced senescent cells in the epidermis.

Ruxolitinib:

Healthspan Benefit: [Xu et al \(2015\)](#) reported that two-month treatment of ruxolitinib in aged 22-month-old mice prevented age-related fat loss, reduced plasma free fatty acids, reduced liver triglycerides, and improved glucose homeostasis.

A1331852 and A1155463

A1331852 and A1155463 were reported to be senolytic in human umbilical vein endothelial cells (HUVECs) and human lung fibroblasts (IMR90 cells) but not in human preadipocytes ([Zhu et al, 2017](#)).

HSP90 inhibitors

Healthspan Benefit: 17-DMAG was reported to be senolytic in human fibroblasts. Intermittent treatment of a mouse model of accelerated aging improved multiple aspects of healthspan (e.g. dystonia, tremor, ataxia, body composition) ([Fuhrmann-Stroissnigg et al, 2017](#)).

Safety: Most senolytics are cancer drugs with potentially serious side effects, though the effects of a 'hit and run' approach are unknown.

Types of Evidence:

- One open-label study of D+Q in idiopathic pulmonary fibrosis

Most of the senolytics currently studied are also anti-cancer drugs that come with serious side effects including neutropenia (low concentration of white blood cells) and thrombocytopenia (low blood platelet count). However, senolytics are given in a 'hit and run' manner, and short-term/intermittent treatment may mitigate some of these effects.

D+Q:

In the idiopathic pulmonary fibrosis trial, most adverse events were mild-moderate in severity. Although it was an open-label trial, side effect profiles were similar to those seen in the placebo arm of previous IPF studies ([Justice et al, 2019](#)).

Quercetin:

Quercetin is a natural flavonol that inhibits PI3K, other kinases, and serpins. It is widely used with few side effects, although the side-effects of long-term use at doses used in senolytic studies is unknown. There are some concerns surrounding *in vitro* mutagenic activity of quercetin, but these effects do not seem to occur *in vivo* in mice ([Harwood et al, 2007](#)). Quercetin is metabolized by the liver, so care should be taken with other first-pass drugs (see [drugs.com](#)).

Dasatinib:

Dasatinib (also known as SPRYCEL) is an inhibitor of multiple tyrosine kinases that is currently marketed by Bristol-Myers Squibb for chronic myelogenous leukemia. Common side-effects include neutropenia, myelosuppression, pulmonary edema, pericardial effusion, pleural effusion, fluid retention, dyspnea, and gastrointestinal hemorrhage ([drugs.com](#)). These side-effects are related to its use as a cancer drug, where the recommended starting dose is 100mg/day. The IPF clinical trial used 100mg/day.



Navitoclax and ABT-737:

Navitoclax (also ABT 263) and ABT-737 are experimental cancer drugs that inhibit anti-apoptotic proteins including Bcl-2, Bcl-XL, and Bcl-w. Common side-effects potentially include neutropenia and thrombocytopenia ([Wilson et al, 2010](#)).

Ruxolitinib:

Ruxolitinib (also known as Jakafi; from Incyte Pharmaceuticals and Novartis) is another anti-cancer drugs that targets the JAK/STAT pathway. Side-effects include thrombocytopenia, anemia, and neutropenia ([see drugs.com](#)).

Other safety information on particular drugs can be found at [drugs.com](#).

Drug interactions:

Drug interactions will depend on the senolytic used. More information can be found at [drugs.com](#).

Additional Information:

Companies developing senolytics:

Unity Biotechnology: developing small molecules senolytics. They have completed a phase 1 safety study with UBX0101. They also recently acquired an alpha-klotho therapy for cognitive decline.

Unity Biotechnology has completed a safety study of UBX0101 in patients with osteoarthritis, though the results are not published ([NCT03513016](#)).

[Oisin Biotechnology](#): Gene therapy with plasmid that encodes a death protein (capase 9 under p16 or p53 promoter) in a lipid nanoparticle; allows you to specifically target a specific type of p16/p53-expressing cells. It is reported to have good biodistribution and is rapidly cleared in mice and non-human primates. Preclinical mouse studies suggest treatment with p53 promoter increases mean lifespan 11.8%, with p16 promoter increased median lifespan 17.6%, when combining the p53 and p16 promoter an increase in lifespan of 20.2%. They reported an increase in bone density over time with combined promoter (conference information). Oisin's first indication will be cancer.

Other companies have entered the senescent space including [Cleara Biotech](#), [Senolytic Therapeutics](#) (a daughter company of [Life Biosciences](#)), [Everon Biosciences](#), FoxBio (joint venture with Antoxerene and Juvenescence), [Rubedo Life Science](#), and [SIWA Therapeutics](#).



Oisin is developing gene therapy programs, SIWA antibodies, and all others small molecules.

Sources and dosing:

Quercetin and fisetin are common supplements available at any vitamin store. Many of the other senolytics are anti-cancer drugs available with a prescription.

Research Underway:

One study is currently being conducted at the Mayo Clinic ([NCT02848131](#)) testing the effect of dasatinib (100mg/day for 3 consecutive days) and quercetin (250mg – 4 times per day – for 3 consecutive days) in patients with chronic kidney disease. The results are not expected until 2021.

Two studies of fisetin are underway. One study ([NCT03325322](#)) is testing fisetin (20mg/kg/day) over two days and examining inflammatory markers and mesenchymal stem cell function in patients with chronic kidney disease. Another study ([NCT03675724](#)) is testing fisetin (20mg/kg/day) over two days in elderly patients and examining inflammatory markers and frailty measures.

Numerous other clinical trials are planned or underway.

Search Terms:

Senolytic, dasatinib, quercetin, ruxolitinib, navitoclax, ABT-737, ABT-263

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