

FDA Approvals of Aging Therapies Have Started: The Motivation for, Status of, & Path to Eliminating Aging

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These slides: <https://tinyurl.com/AgingPipeline26> or QR:

<https://www.linkedin.com/in/karl-r-pfleger/>
<https://x.com/KarlPfleger>

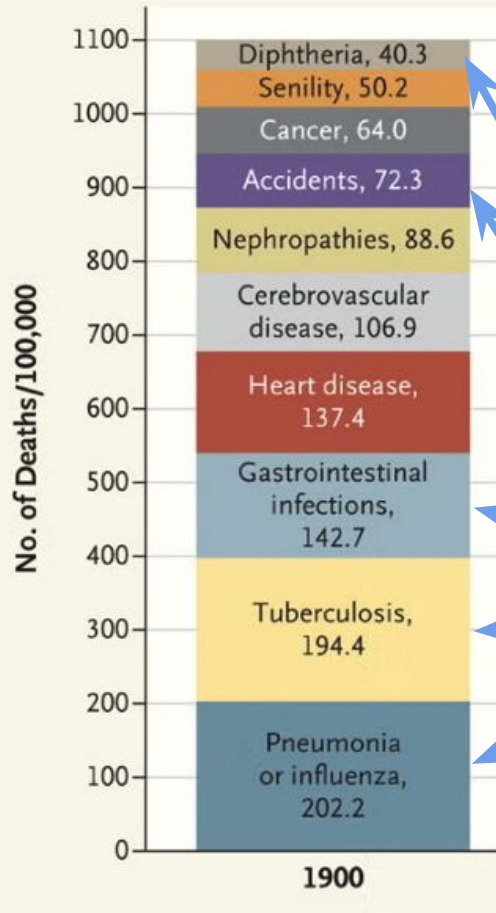
(Investments disclosed on LinkedIn.)

Background: motivation

(9 min)

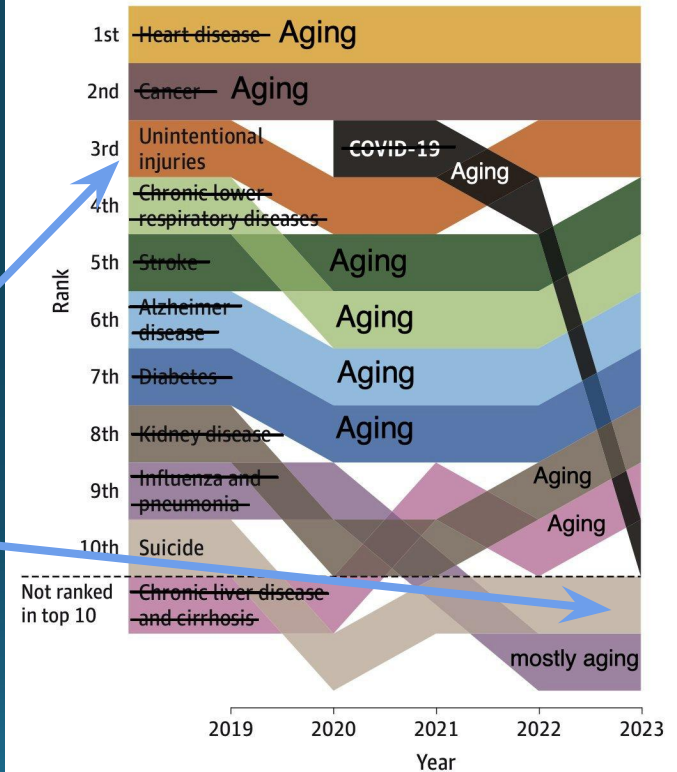
Top causes of death in 1900 vs. now

not primarily caused by aging (at that time)



1900: half of top 10 not age-related, including the **top 3 (all infectious)**

Figure. Trends in the Ranking of Leading Causes of Death—US, 2019-2023



now: 9 of top 10 age-related (suicide dropping to #11), with **many major infectious diseases largely eliminated**
This talk is about doing the same to aging: largely eliminating it.

Simplified capsule history of medicine

antiquity: mystical disease explanations & made up theories (eg “bad humors”)

pre-modern: germ theory, microscopes, chemistry

modern: therapies created by scientific research that discovers how symptoms relate to disease-specific biology/chemistry, for which interventions are developed

With a few exceptions, **medicine reactively treats diseases after they start** (& even in cases where medicine is preventative (vaccines, statins), **most treatments are specific to 1 disease**)

Scientific understanding often proceeds backwards along causal chains:



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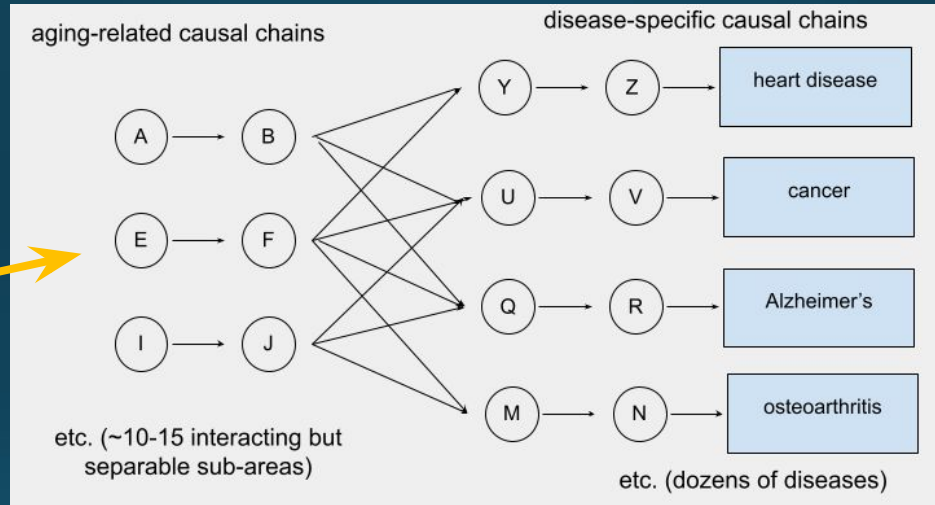


The geroscience ~~hypothesis~~ paradigm

Backward causal science eventually found shared causes of multiple diseases

interfering with any of these can help mitigate multiple age-related diseases

The “Geroscience Hypothesis”



(too many people in the general population still unaware of this!)

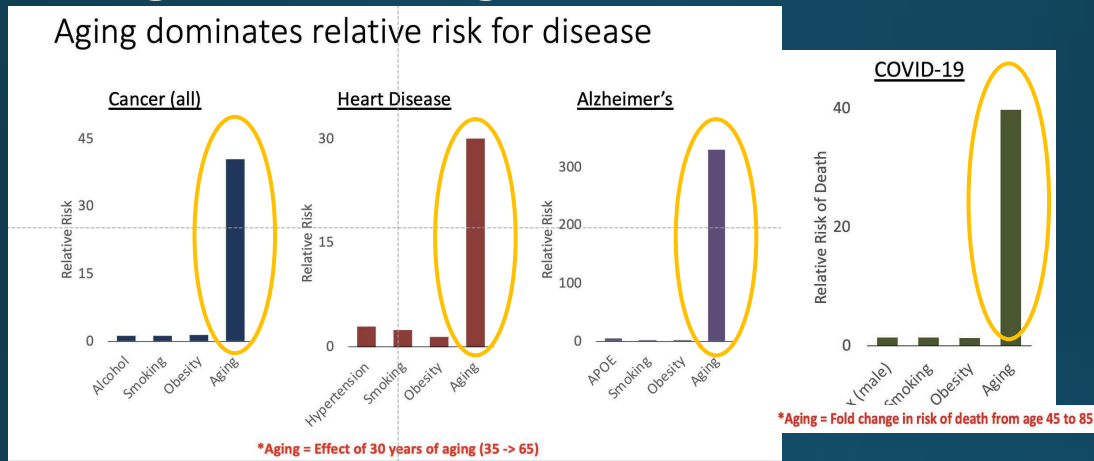
(the north star of the field, *but often forgotten even in the field*)

Growing evidence for several decades now,
but only recently (late 2010s) did an aging biotech industry really get going.

Now that it's clear we can interfere with aging itself, it makes clear a:

Huge funding misallocation

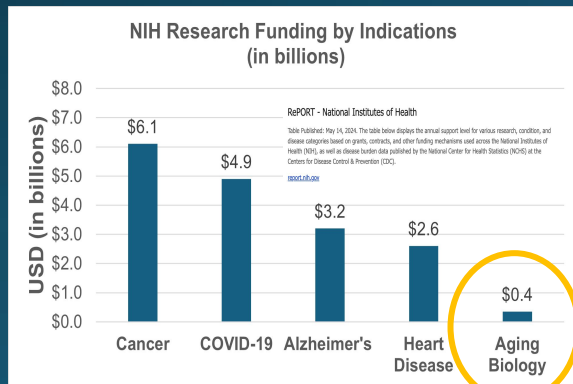
what
causes bad
diseases &
deaths



Geroscience motivations

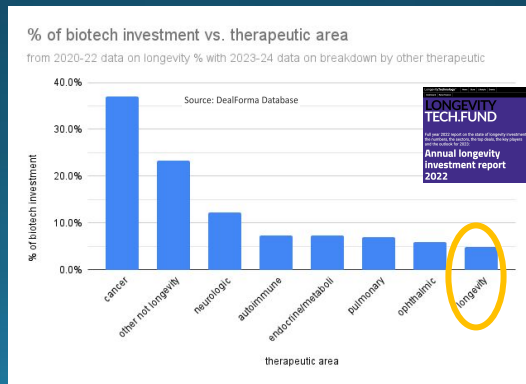
Aging causes 90% of deaths in developed countries,
70%+ globally,
>100,000 deaths/day
(=33 Sept 11 attacks/day,
1-2 Covid pandemics /yr or
WW2 every 2-2.5yrs)

govt: aging <1% of NIH; cancer gets 10x



how
society
allocates
R&D \$

venture: aging ~5%, cancer gets ~7.5x



Philanthropic:
aging gets small %;
cancer gets 10x+

societal 1yr delay in
aging worth \$38 trillion
(Scott et al, 2021)

Huge funding misallocation

NIH gives only \$1/year per American to basic aging research

Aging research gets 80x less per death than cancer research gets

US research funding vs deaths, by cause

NIH research funding of a disease or cause of death in 2024, plotted against annual US deaths from that cause in 2023. Ageing causes over 80% of US deaths, but receives just 0.8% of NIH funding.

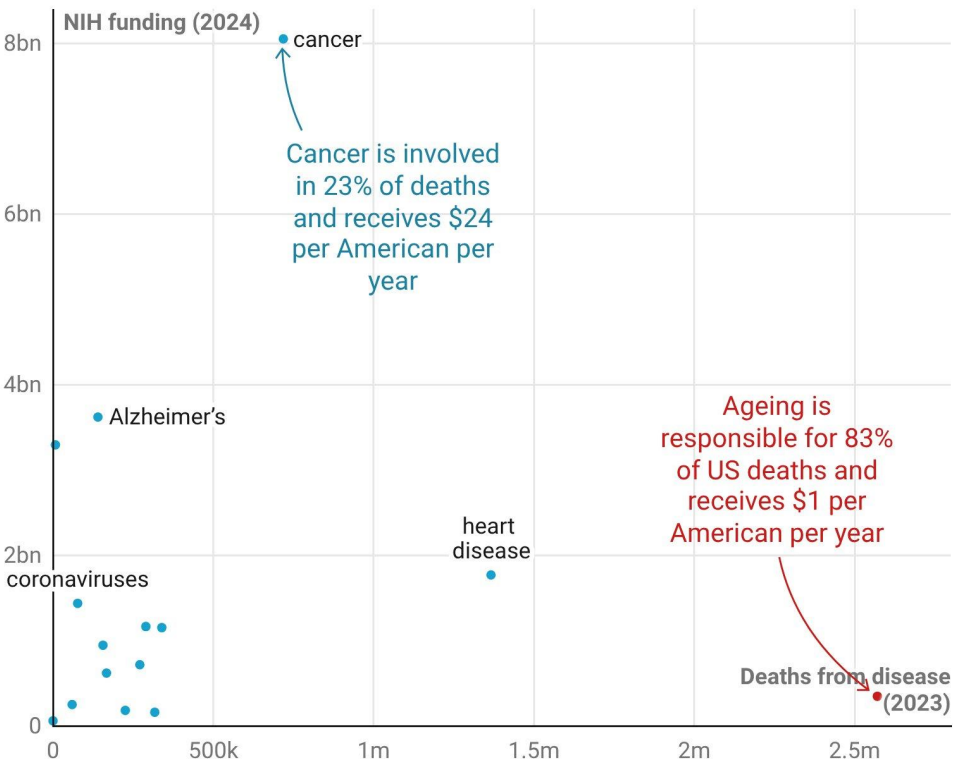


Chart: The Longevity Initiative • Source: RCDC Funding Survey / CDC / analysis by The Longevity Initiative • Created with Datawrapper

Common objections rebutted

Previous slides covered motivations.

There are also objections to the field, but these also have good rebuttals.

[New preprint](#) & upcoming book chapter:

Eliminating Aging:

Motivations, Objections and Counterarguments

Karl Pfleger^{1*}

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Abstract

Adult biological aging causes the majority of human death and arguably human suffering. Scientific advances suggest that humanity can directly mitigate aging's harms and possibly eliminate them entirely, with compelling arguments for doing so, both common-sense arguments and quantitative analyses. However, most of the public and even some in the field are unaware of many of these arguments. In addition, many people intuitively think increased longevity will cause several problems. We review the arguments for mitigating or eliminating aging and show that each of 11 common objections has multiple strong counterarguments. We introduce seven categories of counterarguments and map 43 objection-specific counterarguments into these. Society does not currently fund research on aging and its treatments commensurate with the harm caused or potential benefits to humanity. This review summarizes the compelling benefits of treating aging and dispels the common myths of anticipated negative side effects.

counterargument category → for objection ↓	unlikely	not a problem	ethical perspective	analogy to cancer	net good	reversal test	longevity might help	relationship to other objections
overpopulation	Depends more on fertility rates, which naturally decrease. Underpopulation is now the worry.	We're not close to an upper limit. Adaptation & efficiency gains are possible & likely.	Living people have more right to (continued) life than people who don't exist yet.	If we shouldn't stop trying to cure cancer on population grounds, same applies to aging.	Aging's harms (death, suffering) outweigh harms from overpopulation.	Aging is one of the worst imaginable methods of population control.	Longer lives may improve overall environmental stewardship as people expect to see the results.	Incompatible with only-for-the-rich.
only for the rich	Costs decrease over time, within a generation. Profit margins spread over billions of people.	Even expensive treatments cost less than elder care, so governments will subsidize them.	Morally wrong to withhold things that save lives even if only some people can have them.	Expensive new cancer therapies are cheered as breakthroughs.	Most wealthy would trade wealth for health, showing benefits exceed even high costs.	Imposing aging on the rich if they already had therapies would likely delay wider access.		Incompatible with overpopulation.
long-lived dictators	Globally, the trend is for fewer dictatorships.	Often, dictator death does not end the dictatorship.		Ending cancer R&D to kill dictators isn't advocated.		Condemning billions to death is the worst overthrow plan.		Related to low job turnover.
boredom	Boredom doesn't generally cause death wishes & suicide.	The world becomes less boring over time.	Killing everyone to prevent boredom in some is unacceptable..			Aging is a much worse cure for boredom than other options.	Aging-induced limitations exacerbate boredom.	
slower progress & idea stagnation	Basic desires & universal human drives spur progress not long term future decline & death.	Going slower when one has more time is a luxury, not a problem to be seen as worse.	We don't kill people for producing fewer ideas or not changing their minds.			Aging is a much worse way to spur ideas & faster progress compared to alternatives.	Longer youthful cognition allows wider & deeper knowledge so may create faster progress.	Related to low job turnover (gerontocracy).
longer decrepitude	The current sick care system extends bad health. Treating aging solves this.		We don't kill the decrepit against their will, as demonstrated by current practice.				Treating aging will reduce or eliminate even the current decrepitude.	Related to unsustainable pensions & healthcare costs.
unsustainable pensions / healthcare costs	Same as directly above. Aging & sick-care cause rising healthcare & pension costs.						Eliminating aging will eventually remove the need for pensions & elder care.	Related to longer decrepitude.
unnatural		Unnatural does not imply bad. Many natural things are bad & unnatural good.		Tumors are natural & CAR-T & other cancer treatments are unnatural.				
missing aging's (or death's) good aspects		Experience, wisdom, & the like result from living not aging.	Even if there's a tradeoff, people should have a choice.				Longer longevity should increase wisdom & related good things.	
low job turnover (gerontocracy)	More skilled workers is good for society. New industries will create new jobs.	Wealth, power, & influence will still go to talented & hard working young.				Forced aging & death is a worse policy than forced job turnover.		Related to slower progress & idea stagnation, and to long-lived dictators.
the future will be worse	Objective stats (poverty, health, violence, leisure, GDP/person, ...) show the world improving.		Some people's assessment shouldn't condemn others who feel better about the world.			Even a future worse than today would be less bad without aging.		

Table 1: For each of the 11 objections to mitigating aging or increasing longevity, the counterarguments are summarized briefly and categorized into one of seven categories. Each cell in this table corresponds to a specific subsection in the text. Each cell here is necessarily very brief. The corresponding full subsection explains subtleties and in several cases cite references.

Most comprehensive/scholarly rebuttals review.

Just a few example details:

Overpopulation is unlikely:

70→∞ lifespan change would only raise population by the same amount as a 10% fertility rise.
Fertility naturally declines with economic growth.
#kids/family dropped >50% since 1970s.
Half of humanity now lives in countries with fertility rates <2.

Even expensive therapies won't only be for the rich:
Lifetime 65+ Medicare + support services ~\$600-700k in 2055.
Govt saves money by subsidizing therapies where needed.
(Social security roughly doubles the numbers.)

Long-lived dictators don't pass the cancer analogy:
No one advocates ending cancer R&D to kill dictators.

Low job turnover fails the reversal test:

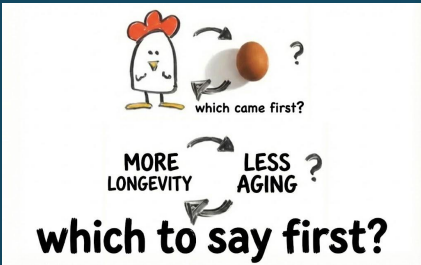
In a world without aging where low job turnover is a problem, creating a pathology that slowly degenerates & tortures everyone for decades before inevitably killing them is a much worse solution than others, e.g.: simple forced job turnover.

[See the chapter coming Sep'26 (or preprint) for references.]

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Longevity medicine isn't about getting more of something. It's about treating a pathology.



Aging is the pathology

Longevity is the *side effect* of treatment



scientific field name	pathology	effect of treating the pathology	NIH institute name	most significant/ cited (conceptual framework) paper	common vernacular	common label is	funding
oncology	cancer	overall survival (or progression-free survival)	National Cancer Institute	Hallmarks of Cancer	cancer	the pathology	high
geroscience (or biogerontology)	aging	longevity	National Institute on Aging	Hallmarks of Aging	longevity	the effect of treating the pathology	low

See [here for more on this](#)

A focus on mitigating aging pathology avoids hand-wringing about lifespan vs. healthspan.

Field Status, especially the late clinical stage pipeline

(10 min)

AgingBiotech.info: everything important in the field

← → ↻ ↺ ⌂	agingbiotech.info/index.html	🔍 ☆ 🌈 📄 ⋮
AgingBiotech.info Home Books Blogs Categories Companies Conferences Databases Diagnostics Forums InvestorsXCompanies Jobs About Contact Journals Motivations Nonprofits Objections Opportunities People Podcasts Therapeutics Trials Videos		
Books	Best books on aging/longevity, objectively ranked	
Blogs	Very small table of the most relevant blogs	
Categories	Table mapping between Hallmarks, SENS, Pillars, & more	
Companies	Big table of (for-profit) aging companies, w/ sub-tabs	
Conferences	Confer	
Databases	Aging o	
Diagnostics	Availab	
Forums	Very sr	
InvestorsXCompanies	~1page	
Jobs	Very sr	
Journals	Aging j	
Motivations	Best m econ	
Nonprofits	Nonpro charite	
Objections	~1page counte	
Opportunities	Misc op	
People	Notable	
Podcasts	Small t	
Therapeutics	Availab eviden	
Trials	Very sr trials	
Videos	The m	

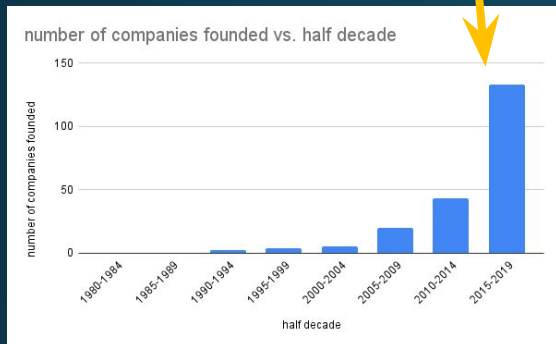


~300 companies + ~200 partial listings

[how to sort/filter] [* = see notes col] [gray = older info]	a.s. aging	aging mission?	core aging area?	multiple aging diseases?	core aging cells?	is it rejuvena- tion?	operating status	short summary (intended to be objective, usually from company website, edited for brevity)	clinical stage (& link to pipeline)	pipeline image (if not clickable, use clin stage link)	Tx or Drx	categories (map to SENS & hallmarks)	diseases/ indications (not comprehensive or canonicalized)	species (beside humans)	clinical trials	targets / pathway / MoAs	modalities	regulatory category	team	geo(s)	public ?
Calico	yes	yes	yes	yes	yes		operating	broad mission to improve health and longevity using technology to advance biological science	ph 2/3 trials (full list of trials)		Tx	big industrial RAD	ALS, cancer, vanishing white matter		10	elF2R		pharma	Arthur D. Levinson, Cynthia Kenyon	SF	no
BioAge Labs	yes	yes	yes	yes	yes	no	operating	human long-term biobanks + multimomics + AI drug platform for metabolic aging w/ APJ & NLRP3 leads	ph 2 trials		Tx	metabolism, inflammation	obesity, muscle atrophy, neuroinflam		4	APJ, NLRP3	drugs	pharma	Kristen Fortney, Eric Morgan, Paul Rubin	SF	yes
Altos Labs	yes	yes	yes	yes*	yes	yes	operating	cell rejuvenation by partial/epigenetic reprogramming; big & academic-like; aiming to be "Bell Labs" of bio umbrella company focused on aging w/ both (ketone) supplement & pharma programs (PAI-1, GDF15, CD38, ketones, & plasmalogens, autophagy, liver & limb regen)	pre-clinical		Tx Drx	epigenetic, big industrial RAD	fibrosis, muscle wasting in cancer, RA, HF, dementia			OSKM PAI-1, GDF15, CD38,	drugs & others, supplement	pharma, nutraceu- ticals	Hal Barron, Rick Klauser, Hans Bishop, Juan Carlos Richard Marshall, Greg Bailey, Jim Mellon	SF, SD, Cambridge	no
Juvenescence	yes	yes	yes	yes	yes		operating	AI for drug discovery platform with particular focus on aging diseases	commercial, ph 2 trials*		Drx	umbrella company	muscle wasting in cancer, RA, HF, dementia			OSKM PAI-1, GDF15, CD38,	drugs & others, supplement	pharma, nutraceu- ticals	Richard Marshall, Greg Bailey, Jim Mellon	London, NYC	no
InSilico Medicine	yes*	yes		yes	yes		operating	AI for drug discovery platform with particular focus on aging diseases	ph 2 trials		Tx	broad platform	IPF, kidneyDs, cancer, Covid			OSKM PAI-1, GDF15, CD38,	drugs & others, supplement	pharma, nutraceu- ticals	Alex Zhavoronkov, Feng Ren	China, UAE, US, Canada	no
Stealth BioTherapeutics	yes	yes	yes	yes	no	no	operating	mitochondria platform for age & genetic mito diseases; first a peptide to stabilize inner mito membrane treat aging, 1st w/ senolytics, esp for eye diseases (these OA failed), also other programs inc. VEGF & bioRxiv (partnered)	ph 3 trials		Tx	mitochondria	AMD, DMD, ALS, mito-neuroDs		29	cardiolipin	peptides, small molecules	pharma	Reenie McCarthy	Boston	no*
Unity Biotechnology	yes	yes	yes	yes	yes	yes	operating acquired*, operating independ	leads from blood proteins that go up or down with age, eg young blood fractions	ph 2 trials		Tx	senescence, klotho	eyeDs, neuroDs		8	Ed-L, VEGF, klotho	small molecules, proteins	pharma	Anirvan Ghosh	SF	yes
Alkermes (owned by Grifols)	yes	yes	yes	yes	yes	yes	operating	leads from blood proteins that go up or down with age, eg young blood fractions	ph 2 trials		Tx	cell communication	AD, PD, wAMD, ESRD, MCI		13	iduronate 2-sulfate, LRRK2,	drugs, biolog	pharma	Eather Rawner, Tony Wyse-Garay	SF	public owner
Denali Therapeutics	yes*	no*	yes	yes	no		operating	drug discovery for neurodegeneration, optimized for brain delivery, guided by genetics & biomarkers, but also lysosomal storage diseases & systemic inflam	ph 3 trials		Tx	autophagy, inflammation	PO, FTD, Hunter's, ALS, AD, MS, inflammation		22	iduronate 2-sulfate, LRRK2,	small molecules, biologics	pharma	Ryan Watts	SF	yes
Life Biosciences	yes	yes	yes	yes	yes	yes	operating	epigenetic reprogramming via OSK and small molecules for chaperone mediated autophagy	pre-clinical		Tx	epigenetic, autophagy	eyeDs, neuroDs			OSK, LAMP2A	gene therapy, small	pharma	David Sinclair, Jerry McLaughlin	Boston	no
Scholar Rock	yes	no	yes	yes	no	no	operating	platform for targeting precursors of growth factors including myostatin & TGFβ	ph 3 trials		Tx	muscle atrophy, cardiometabolic, cancer, fibrosis		6	TGFβ	monoclonal antibodies	pharma	Jay Backstrom	Boston	yes	
Immunka Inc.	yes	yes	yes	yes	yes	yes	operating	stem-cell secretome products for age-driven immune dysfunction, muscle regeneration & metabolism	ph 2a trials		Tx	stem cells	cardiometabolic, cancer		1	AMPK, mTOR	secretome cocktail	pharma	Hans Kleinsteid	LA	no
Cambrian Bio	yes	yes	yes	yes	yes		operating	umbrella company focused on aging w/ leads in AMPK, mitochondria, mTOR, & more	ph 1b trials*		Tx	umbrella company	cardiometabolic, cancer		0*	AMPK, mTOR	drugs	pharma	James Peyer	NYC	no
Juvena Therapeutics	yes	yes	yes	yes	yes	yes	operating	computationally-driven platform based on human stem cell secreted proteins engineered into novel biological	pre-clinical		Tx	stem cells, cell communication	DM1, obesity, fibrosis, OA			MLASCA, CAR-T, NK	biologics	pharma	Hanadi Yousef, Jeremy O'Connell	z	no
Cellularity	yes	yes	yes	yes	no	yes	operating	allogeneic placental-derived cell therapies (MLASCA, CAR-T, NK cells) for aging, degenerative, immune diseases, cancer, also biomaterials	ph 1/2 trials		Tx	stem cells / allogeneic	cancer, muscleDs, autoimmuneDs		17	cells	cell therapy	pharma	Robert Hariri	NYC	yes
Mesoblast	yes	no	yes	yes*	no	yes	operating	allogeneic mesenchymal stem cells for multiple (esp inflammatory) diseases	ph 3 trials*		Tx	stem cells / allogeneic	HF, deg-disCd, Covid (& non-agedDs)		42	MSCs	cell therapy	pharma	Silvia Rescu	NYC, Melbourn	yes
Rubedo Life	yes	yes	yes	yes	yes	yes	operating	target pathological cells that drive aging, esp senescent cells, using AI, single cell seq, & precision targeting	pre-clinical		Tx	epigenetic	AD, psoriasis, IPF, NASH, cancer,			small molecules	pharma	Maxine (Ph.D.)	SF	yes	

Scope is effectively
things that embody the
geroscience hypothesis
(as primary focus)

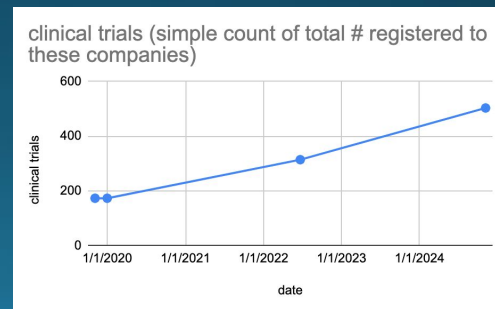
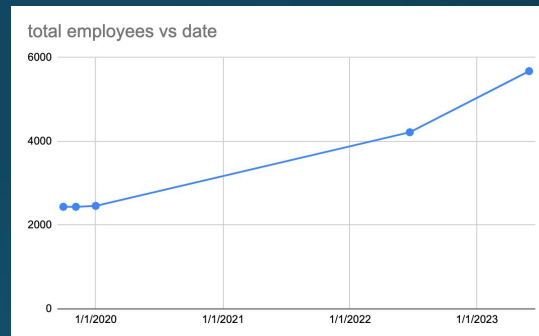
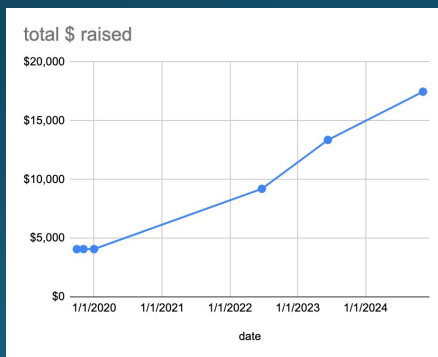
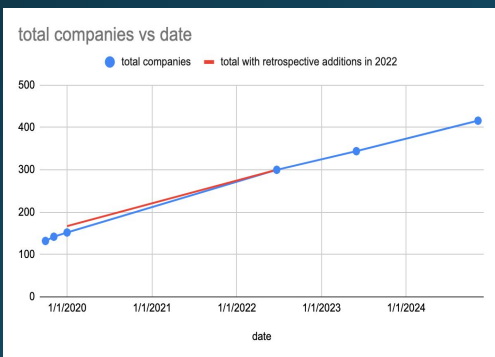
Aging biotech started in earnest late 2010s...



Then **steady growth** & clinical progress for 5 years, but **not yet exponential**

(as of 2024 Nov)

Data from [AgingBiotech.info](https://agingbiotech.info).



company	1/1/2020	most advanced trial phase	11/5/2024
Alector	2	✓	3
Alkermes	2		2
Athersys	3		3
BioAge	pre-clinical	✓	2
Biophytis	2	✓	3
Bioquark	1	defunct	
Biosplice (was Samumed)	3		3
Calico	pre-clinical	✓	3
Cerevance	1	✓	3
CohBar	1		1
Denali	1	✓	3
Eidos	3		3
GEN1E			
Lifesciences	pre-clinical	✓	2
Genome Protection	2	defunct	
GenSight	3		3
InSilico Medicine	pre-clinical	✓	2
Intervene Immune	pre-clinical	✓	2
Longeveron	2		2
Longevity Biotech	1		1
LyGenesis	pre-clinical	✓	2
Mesoblast	3		3
Minovia	2	defunct	1
Navitor	1	✓	2
Pentraxin	2		2
Pharmatrophix	2		2
Proclara	1		1
Proteostasis Tx / Yumantia	2	defunct	
resTORbio	3	defunct	
Retrotope	3	defunct	
Stealth BioTherapeutics	3		3
Vaxxinity (was United Neuro)	2	✓	3
Unity Bio	1	✓	2
regressed or failed			6
progressed			13
progressed by 2+ steps			6
defunct			0
pre-clinical			6
	1		8
	2		10
	3		8
total	32		32

Not shown but happening: conferences increasing, journal impacts increasing, media coverage, etc.

Poll:

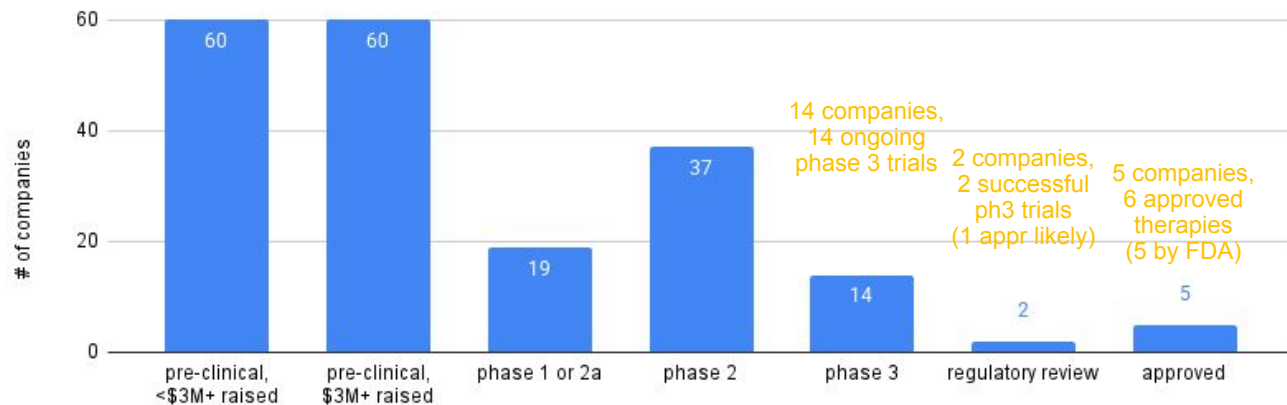
How long do you think it until aging is largely eliminated
as a cause of death & driver of chronic disease?

The next 5-10yrs look exciting

(as of 2025 Nov)

of companies vs. clinical status

(only not defunct/aborted companies in pharma regulatory category)



	pre-clinical <\$3M+ raised	pre-clinical, \$3M+ raised	phase 1 or 2a	phase 2	phase 3	regulatory review	approved
success rate to approval	1.5% ?	4.5% ?	10%	15%	55%	90%	100%
better for non-onco trials			14%				
expected approvals, based on 1 trial per company	1	3	3	6	8	2	6
expected approvals based on counting # individual trials that still have a chance of success					8	2	6
avg time left to approval	8-9yrs	7-8yrs	4.5-5.5yrs	3-4yrs	2-3yrs	<1yr	0

This omits:
supplements,
diagnostics,
veterinary, &
trials from companies not aging focused
(so underestimates)

← increasing uncertainty

[\[List of approvals & ph3 trials\]](#)

Ph.3 trials MoAs generalize

- 3 approved: **ATTR** treatments (5 if also including from non-aging companies)
- 1 approved (3 inc. Asia) + 4 trials: **stem cells** (most allogeneic)
- 1 (accelerated) approved + 1 trial: **mitochondria** MoAs (cardiolipin, ND4)
- 1 met ph3 endpoint (but approval delayed) + 1 trial: anti-**myostatin** mAb
- 1 met ph3 endpoint (but switching indications): **MAS** receptor (affects mTOR & AMPK)
- 3 trials: **FGF21** mimetic
- 1 trial: **NLRP3** (inflammation: IL-1 β & IL-18)
- 1 trial: **ISR**—integrated stress response (proteostasis)
- 1 trial: **PGRN**—progranulin
- 1 trial: **GPR6**

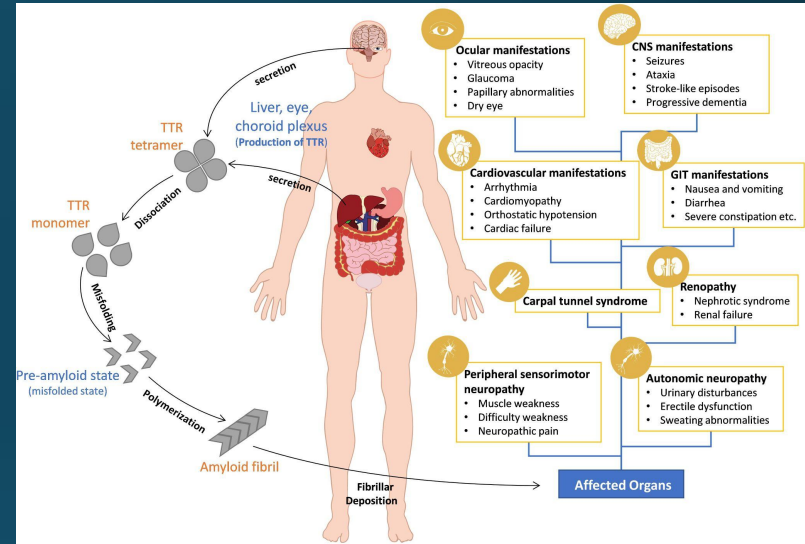
These should all be relevant for diverse age-related diseases, embody the geroscience hypothesis.
This is quite different from most of prior biopharma.

Ph3 trials: AgingBiotech.info/trials top row link

approval status / stage	trials (& link)	# ph3 trials w/ chance of success	company	phase	indication (disease)	indication age related?	target / pathway / MoA [colors in these columns show commonality of target/MoA]	potential for label expansion or off-label prescribing for multiple age-related diseases?	intervention [colors show commonality]	study completion	FDA approval date	ClinicalTrials.gov ID	as of	notes
approved	5 ph3 trials, inc ACT-EARLY		Eidos/BridgeBio	approved	hATTR, ATTR, ATTR-CM	yes, some	ATTR (Transferrin Amyloidosis), TTR stabilizer	yes, implicated in several areas (also hearing loss)	acoramidis (Attruby)		2024-11-22		2025-02-07	1 of several approved ATTR tx, inc. (not in this table) Pfizer's tafamidis (Vyndaqel or Vyndamax) & lonis's inotersen (Tegsed).
approved	3 ph3 trials, inc HELIOS-A, HELIOS-B		Amylam	approved	hATTR, ATTR, ATTR-CM	yes, some	ATTR (Transferrin Amyloidosis), RNAi TTR suppressor	yes, implicated in several areas (also hearing loss)	utrisiran (Ammvutra)		2022-06-13		2025-02-07	1 of several approved ATTR tx, inc. (not in this table) Pfizer's tafamidis (Vyndaqel or Vyndamax) & lonis's inotersen (Tegsed).
approved	3 ph3 trials (3 completed), inc APOLLO, HELIOS-A		Amylam	approved	hATTR, ATTR, ATTR-CM	yes, some	ATTR (Transferrin Amyloidosis), RNAi TTR suppressor	yes, implicated in several areas (also hearing loss)	patisiran (Onpatro)		2018-08-10		2025-02-07	1 of several approved ATTR tx, inc. (not in this table) Pfizer's tafamidis (Vyndaqel or Vyndamax) & lonis's inotersen (Tegsed).
approved	approved 2012 in S. Korea		Medipost	approved	OA (knee)	yes	cord-blood-derived MSCs	yes, for a wide range of age-related diseases	CARTISTEM		2025-09-11			
approved	3 ph3 trials (all completed)		Mesoblast (remestemcel)	approved	GVHD	no	allogeneic mesenchymal lineage stem cells	yes, for a wide range of age-related diseases	renesemcel-L (Ryocil, was called Prochymal)		2024-12-18		2025-02-13	
accelerated approval	4 trials (2 completed)	1	Stealth Bio/Therapeutics	1 regulatory review, 2 ongoing ph3	Barth Syn, prim mlo dis's, dry AMD	no	cardiolipin on inner mitochondria membrane, to stabilize membrane, JATP, JRCOS	yes, for a wide range of age-related diseases	elamipretide (Forzynta), a peptide also known as SS-31 (used for years by biohackers)				2025-10-01	Sep'25 update: FDA granted accelerated approval for Barth Syndrome. May'25 update: FDA rejected approval for Barth after positive phase 3 data & their own advisory committee voted to approve. But Stealth still pursuing Barth accelerated approval & phase 3 trials for the other 2 indications still ongoing. More articles & discussion.
successful phase 3 results	SAPPHIRE	1	Scholar Rock	regulatory review	SMA	no	anti-myostatin monoclonal antibody	yes, should help with broad muscle conditions, age-related atrophy, frailty, sarcopenia*	apitegromab (mAb)	2024-12	NCT05156320	2024-12-05		Trial succeeded, met primary endpoint (reduced incidence respiratory failure or death) 44%, significantly, press release, academic paper. Trial succeeded, met primary endpoint (news article, academic paper). Approval delayed by unrelated manufacturing issues at Novo Nordisk run plant. *And already in phase 1 by Scholar Rock for obesity.
successful phase 3 results	COVA	1	Biophlys	regulatory review	Covid-19 (soon sarcopenia ph3 to start, see notes*)	yes	activate MAS receptor (component of RAS system, affects mTOR & AMPK) to increase resilience	yes, for a wide range of age-related diseases (& in trials for sarcopenia & obesity)	BIO101 / Ruvemri (a SAS1 inhibitor) (formerly called Sarcomene?)	2022-09	NCT04472728	2024-12-06		Strange set of trials in which early trials treated 1 eye using other as control but control eye vision improved too, leading to failure relative to "control" despite improvement. 5 year followup data showed sustained vision improvement. Company still seems to be pursuing & considering it to be phase 3 per pipeline.
phase 3	3: REVERSE, RESCUE, RESTORE & REFLECT	1	GenSight Biologics	3	LHON	no	milo ND4 gene allotypic expression w/ miloc targeting of mRNA to milo membrane	yes as part of more allotypic expression of milo genes; unclear for just for ND4	GS010 / Lumevoq (gene therapy)	2018-12 to 2022-07	NCT02652780, NCT034065767, NCT03408104, NCT03293524	2025-02-18		
phase 3	ONVX	1	Scholar Rock	3	SMA	no	anti-myostatin monoclonal antibody	yes, should help with broad muscle conditions, age-related atrophy, frailty, sarcopenia	apitegromab (mAb)	2027-01	NCT05626855	2024-12-05		Follow-up trial to their other SMA trials.
phase 3	SYNCHRONY: 3 NASH/MASH related trials	3	Akera Therapeutics	3	NASH/ MASH	yes	EGF21 mimetic	yes, for a wide range of age-related diseases	Eltusfermin (EFK) (biologic protein)	2026-10 to 2032-11	NCT06161571, NCT06528314, NCT06215716	2024-12-06		
phase 3	INFRONT-3	1	Alector	3	FTD (GRN)	yes	progranulin (PGRN), a key regulator of immune activity	yes, based on progranulin relation to immune function & lysosome function & multiple dementias	ALD01 / Latozinemab (mAb)	2027-10	NCT04374136	2024-12-06		See also continuation study NCT06111014.
phase 3	TransportNPC	1	Cycle Therapeutics	3	Niemann-Pick Disease	no	cyclodextrin for cholesterol transport in place of NPC1	yes, and also in phase 2 for AD by Cycle (& diff cyclodextrin pursued for CVD by Cylarity)	Trappsol Cyclin (hydroxypropyl beta cyclodextrin)	2026-06	NCT04860960	2024-12-06		
phase 3	CVN424 for PD trial	1	Cerevance	3	PD	yes	GPR6	yes, GPR6 thought to be a target for several dementias	Solengepras / CVN424	2026-03	NCT06553027	2024-12-06		
phase 3	4 ph3 trials (3 completed, 1 failed)	2	Mesoblast (not remestemcel)	3	disease	yes, some	allogeneic mesenchymal lineage stem cells	yes, for a wide range of age-related diseases	allogeneic mesenchymal lineage stem cells	[various]	NCT02032004, NCT01854567, NCT02412735, NCT06325566	2025-02-08		NCT02032004 failed. NCT01854567 prob failed: its cancer indications not on the pipeline. NCT02412735 ended 2021: had some positive results & in 2023 got RMAT designation, but doesn't seem to be in regulatory review? So call it a ph3 for now.
phase 3	SCUpTOR trial (AusNZ)	1	Cynata Therapeutics	3	OA (knee)	yes	allogeneic MSCs	yes, for a wide range of age-related diseases	allogeneic MSCs		U1111-1234-4897 (ANZCTR)	2025-09-11		Ph3 trial in Australia / New Zealand's Clinical Trials Registry (ANZCTR).
phase 3	Dapansutrile for Acute Gout Flare	1	Ciatec	2/3	gout	yes	inhibit NLRP3 to reduce inflammation by reducing cytokines IL-1β and IL-18	yes, with several other clinical trials from Ciatec for several age-related diseases	QLT1177 / Dapansutrile (small molecule)	2025-10	NCT05658575	2024-12-06		
phase 3	MASTERS-2	1 but paused	Athenrys Inc	3	ischemic stroke	yes	allogeneic Multipotent Adult Progenitor (stem) Cells	yes, for a wide range of age-related diseases	MultiStem (allogeneic MAPCs)	2023-06	NCT03545607	2024-12-06		Planned interim analysis showed study size insufficiently powered to achieved significance, so enrollment paused for further analysis by company. Company pipeline on website says enrollment ongoing.
phase 3	MACoVIA	1 but paused	Athenrys Inc	2/3	ARDS (Covid19)	yes	allogeneic Multipotent Adult Progenitor (stem) Cells	yes, for a wide range of age-related diseases	MultiStem (allogeneic MAPCs)	2023-12	NCT04367077	2024-12-06		Paused pending funding per website pipeline.
phase 3	VISTA-1 & VISTA-2	0	Miletech	3	dry eye syndrome	yes	cardiolipin to inhibit peroxidation in mitochondria	yes, for a wide range of age-related diseases	Visomitin / SKQ1 (small molecule eye drops)	2019-02 & 2020-10	NCT03764735 & NCT04206020	2025-02-16		VISTA-1 succeeded but rather than leading to approval, led to VISTA-2. VISTA-2 met 1 primary endpoint (staining) but seemingly not the 2nd (discomfort). After that 2021 press release talked about a VISTA-3 trial, there doesn't seem to be such a trial & a 2022 press release says Miletech sold SKQ1 rights for dry eye to Essex Bio, but Miletech continues to pursue SKQ1 for other indications.
phase 3	NurOwn trial for ALS	0	Brainstorm Cell Therapeutics	3	ALS	yes	autologous bone marrow derived mesenchymal stromal cells (MSC) that secrete more neurotrophic factors	yes, for a wide range of age-related diseases	debamastocel (NurOwn, MSC-NTF cells)	2020-09	NCT03280056	2025-02-17		Failed, but now planning a phase 3b trial (though not yet using clinicaltrials.gov).
phase 3	hip fracture muscle injury	0	Pluri Inc (prev Pluristem)	3	hip fracture	yes	allogeneic placenta-derived, mesenchymal-like adherent stromal cells	yes, for a wide range of age-related diseases	PLX-PAD (allogeneic mesenchymal-like stromal cells)	2023-09	NCT03451916	2024-12-06		Failed but did demonstrate muscle strength improvements. (See also here.) And product in phase 1 for knee OA, though company no longer has hip fracture as indication in its pipeline.
phase 3	TREASURE	0	Athenrys Inc	2/3	ischemic stroke	yes	allogeneic Multipotent Adult Progenitor (stem) Cells	yes, for a wide range of age-related diseases	MultiStem (allogeneic MAPCs)	2023-02	NCT02961504	2024-12-06		Failed.

ATTR approvals

- At least **5 approvals in recent years** (inc. by Alnylam & Eidos/BridgeBio)
- ATTR affects many areas with aging
- Thought to kill majority of 110+yo's
- Treatment may extend maximum human lifespan
- Classic misfolded protein pathology, but important outside the brain
- Increasingly thought underdiagnosed; increasing evidence in younger seniors
- [More details \(X thread\)](#)
- Why isn't this discussed more in the field?



Stem cell approvals

- stem cells no longer only a wild west of iffy clinics & medical tourism
- 1st mass-producible, off-the-shelf (allogeneic) HSC tx FDA approved 2023
- 1st mass-producible, off-the-shelf MSC tx FDA approved 2024 (Mesoblast)
- 1st MSC any-country-approval 2012! (Korea: 30k+ treated. Good 5-7yr followup. First disease modifying OA tx! Japan ph3 met all endpoints & approval expected. US ph3 started mid-2026.)
- HSCs + MSCs covers a large fraction of human cells
- Narrow indications but many more in trials
- [More details \(X thread\)](#)
- Why isn't this discussed more in the field?

Minor 2026 update: Japan conditionally approved allogeneic iPSC dopaminergic tx for Parkinson's & cardiomyocyte tx for heart failure. Mostly these are approvals for real world pivotal data trial, but they are based early on safety+efficacy trial data.

4 more successful phase 3 trials

Stealth Bio: cardiolipin mitochondria peptide elamipretide aka SS-31 got **accelerated approval** for Barth (2025) & in 2 other phase 3s (dAMD, nPMM)

Scholar Rock: anti-myostatin mAb apitegromab **ph3 met primary endpoint** for SMA (approval delayed: manufacturing issue at Novo run plant); in ph1 for 2nd indication

Biophytis's MAS receptor drug **met primary ph3 endpoint** for Covid but company pursuing new ph3 for sarcopenia before trying for approval

(as noted) Medipost's Cartistem MSCs for knee OA met all ph3 primary & 2ndary endpoints (in Japan; already approved in Korea & ph3 in US just started)

Aggregate pipeline outlook summary

- 6 approvals so far & expect 10 more within a few years, then steady trickle
- Label expansion possible after approvals (how long is a big question)
- All lower bounds since this is only aging-focused biotechs (others have additional assets, eg GLP-1s, other ATTR drugs)
- Uncertainty 5+yr out (current pre-clinicals, esp. w/ growing # new companies)
- Big extra upside & downside surprise uncertainty:
 - Regulatory/funding changes speed trials/research (A4LI proposals: multi-disease designation & disease-burden-based R&D funding)
 - Staff cuts slow FDA or NIH cuts dry up early research feeding pipeline?
- Clinical implications: Likely will be a long time where quality of off-label prescribing, knowing who needs what MoAs (& how to dose), matters a lot.

Why this is important summary

1. One of the things that will help trigger a public opinion sea change & funding
2. When funding rises, it's expansion of this pipeline that will embody progress
3. If \$ don't go 10x, **this that will continue to make steady progress anyway**
(slower than if funding goes 10x, but **still better than the sick-care model**)

Surprising this isn't discussed more (esp. relative to some stuff that is):

- **Different from rapa, metformin, SGLT2**, D+Q, supplements in being on-patent so there are financial incentives to run large trials
- **Different from GLP-1s** in that mostly MoAs likely effective even absent obesity
- **Bryan Johnson** fully expects to live many more decades but mostly discusses what he uses that's avail now & AI but not what's coming soon in biotech.
- Most **Peter Attia** clients will live 2+ decades but he discusses mostly available lifestyle, supplements, & drugs & ignores what's coming soon in the pipeline.

Paths to eliminating aging: the different longevity fields

(12 min)

See [The 2 Longevity Fields](#) for more details.

THE 2 LONGEVITY FIELDS

or 3

(more distant cousin)

*(dominant
subfield)*

*(pursued but not much
recognized)*

whole-body replacement

(brain transplants)
+ brain rejuvenation or
incremental replacement

**broadly
slowing aging**

**divide-and-conquer
rejuvenation**

parts of aging
addressed:

most/all

primarily 1 specific sub-pathology

most/all

effect size on affected
sub-pathologies:

small (slow small %)

up to a total fix (yrs of reversion, repeatable)

total fix

targets are:

processes

stable structures

stable structures

dosing:

continual

infrequently intermittent

infreq. intermittent

Part vs. whole & How much is the goal?



Aging vs. longevity or geroscience hypothesis vs. lifespan extension

Many different views of the field. See [What counts as a longevity drug.](#)

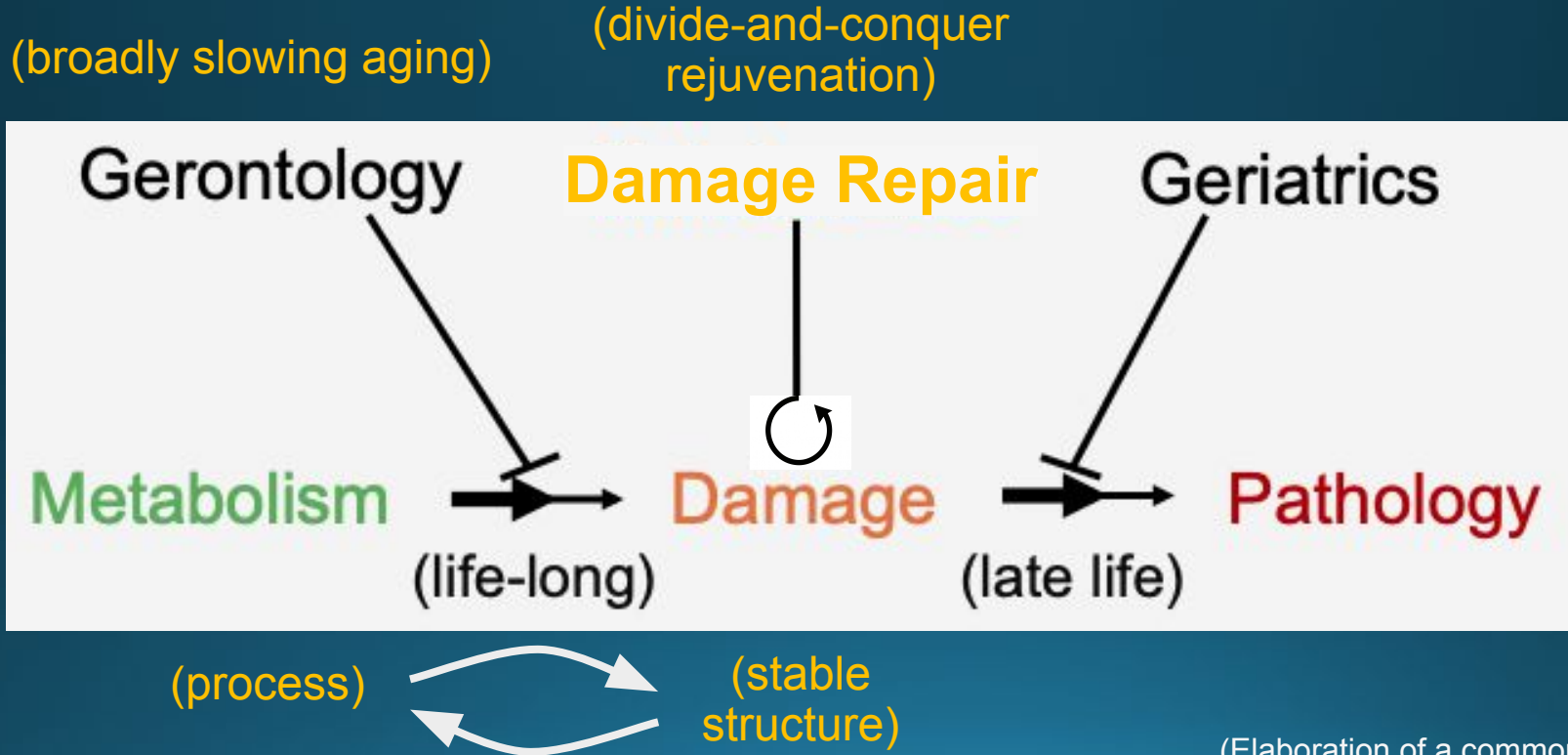
Some aging therapies may not extend lifespans by themselves.

That doesn't mean they don't embody the geroscience paradigm.

Monotherapy lifespan extension isn't a litmus test for the whole field.

All aging sub-pathologies eventually need therapies. Covering more of them is a type of progress of the field. The field should start measuring progress this way!

Processes vs. stable structures



(Elaboration of a common SENS diagram from Aubrey de Grey.)

Aging can be divided into ~ 7–15 separable sub-pathologies

No universal consensus on the best breakdown (eg, Hallmarks vs. SENS)

But many of them are widely agreed on.

See table or AgingBiotech.info/categories for the best simple mapping I'm aware of.

See [de Grey \(2023\)](#) for the most direct published compare/contrast.

7 SENS damage types (2002 & 2003)	9 Hallmarks of aging (2013) or 14 (2022) or 12 (2023)	7 Pillars of aging (2014)	primarily stable structures or mostly only processes
cell loss & atrophy	stem cell exhaustion	stem cell regeration	primarily stable structures
cell senescence	cellular senescence		primarily stable structures
nuclear mutations / cancer	genomic instability	macromolecular damage	primarily stable structures
extracellular crosslinks / matrix stiffening	altered mechanical properties (new hallmark)		primarily stable structures
intracellular junk			primarily stable structures
	telomere attrition		primarily stable structures
	epigenetic alterations	epigenetics	primarily stable structures
	microbiome disturbance / disbiosis (new hallmark)		primarily stable structures
extracellular junk	loss of proteostasis	proteostasis	mostly stable structures
mitochondrial mutations	mitochondrial dysfunction		some clear stable structures
	deregulated nutrient sensing	metabolism	primarily processes
	altered intercellular communication		primarily processes
	disabled macroautophagy (new hallmark)		primarily processes
	(chronic) inflammation (new hallmark)	inflammation	primarily processes
	splicing dysregulation (new hallmark)		primarily processes
		stress	primarily processes

How big is the problem of addressing all aspects of aging?

The next few slides give a quick sense by showing the aggregate clinical pipeline (including earlier stages) for just the divide-and-conquer rejuvenation portion of the field.

Majority of the field focuses on processes & slowing aging

Stable structure targets are (inadvertently?) de-emphasized.

ITP Top 10 for lifespan extension: All primarily target process, not stable structure.

Rapamycin: ~26/23% (F/M)
 Acarbose: ~22/5% (M/F)
 17- α -estradiol: ~19% (M)
 16 α -hydroxyestriol: ~15% (M)
 Canagliflozin: ~14% (M)
 Astaxanthin: ~12% (M)
 NDGA: ~12% (M)
 Halofuginone: ~9% (M)
 Mitoglitazone: ~9% (M)
 Aspirin &
 Meclizine (*Tie*): ~8% (M)

7 SENS damage types (2002 & 2003)	9 Hallmarks of aging (2013) or 14 (2022) or 12 (2023)	7 Pillars of aging (2014)	primarily stable structures or mostly only processes
cell loss & atrophy	stem cell exhaustion	stem cell regeration	primarily stable structures
cell senescence	cellular senescence		primarily stable structures
nuclear mutations / cancer	genomic instability	macromolecular damage	primarily stable structures
extracellular crosslinks / matrix stiffening	altered mechanical properties (new hallmark)		primarily stable structures
intracellular junk			primarily stable structures
	telomere attrition		primarily stable structures
	epigenetic alterations	epigenetics	primarily stable structures
	microbiome disturbance / disbiosis (new hallmark)		primarily stable structures
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	deregulated nutrient sensing	metabolism	primarily processes
	altered intercellular communication		primarily processes
	disabled macroautophagy (new hallmark)		primarily processes
	(chronic) inflammation (new hallmark)	inflammation	primarily processes
	splicing dysregulation (new hallmark)		primarily processes
		stress	primarily processes

Nir Barzilai & co. top 12 ranking: All but 1 target process.

An Updated Prioritization of Geroscience-Guided FDA-Approved Drugs Repurposed to Target Aging

Michael Leone^{1,2}, Nir Barzilai^{1,2*}

Gerotherapeutic	Hallmarks of aging	Preclinical healthspan	Preclinical lifespan	Human healthspan	Human mortality	Score (out of 12)
SGLT2 inhibitors	2	2	2	3	3	12
Metformin	2	2	1	3	3	11*
Bisphosphonates	2	2	1	3	3	11
GLP1 receptor agonists	2	2	0	3	3	10*
Acarbose	2	2	2	3	0	9*
Rapamycin	2	2	2	3	0	9
Methylene blue	2	2	2	3	0	9
ACE inhibitors/ARBs	2	2	1	3	0	8
Dasatinib + (quercetin)	2	2	1	3	0	8
Aspirin	2	2	2	1	0	7
Beta blockers	1	2	1	0	3	7
N-acetyl cysteine	2	2	1	0	0	5*

Table 1 Ranking of FDA-approved drugs as potential gerotherapeutics based on scoring (out of 12) for preclinical and clinical evidence. Evidence suggesting COVID-19 mortality benefit is delineated by an asterisk in the total score column.

Stable structural aging subpathology subcategories & why important (related diseases)

structural subpathology category label [not exhaustive]	subcategories [not exhaustive]	diseases caused/exacerbated [not exhaustive]
(long-lived) senescent cells	senescent: fibroblasts, endothelial cells, glial cells, preadipocytes, immune cells	IPF, OA, cancer, CKD, CVD, T2D, AD, PD
non-senescent stable pathological cells	exhausted/anergic T-cells, CHIP, persistent myofibroblasts, hypertrophic/inflamed adipocytes	immunosenescence, CVD, fibrotic diseases, T2D
extracellular matrix remodeling & stiffening	AGEs & collagen crosslinking, elastin fragmentation, vascular calcification, lens crystallin crosslinking	diabetic retinopathy, hypertension, CVD, aneurysms, CKD, skin aging, presbyopia
protein misfolding & aggregates	ATTR, α -synuclein, tau, A β	HF, neuropathy; dementias: PD, AD, Lewy body dementia
intracellular toxic molecules	lipofuscin, 7-ketocholesterol, unesterified free cholesterol	atherosclerosis, AMD, AD, PD
cell loss / tissue atrophy & stem cell degradation	neural stem cell depletion, muscle satellite cell degradation, thymus involution, cartilage loss, bone degradation, beta-cell loss	T2D, sarcopenia, cognitive decline, immunosenescence, OA, osteoporosis
mitochondria DNA mutations	4977-bp mtDNA deletion, mitochondria clonal expansion	sarcopenia, PD, presbycusis, HF
nuclear DNA changes	cancer mutations, non-malignant clonal expansion (inc. CHIP), telomere shortening	cancer, atherosclerosis, all senescent cell diseases (IPF, CKD, OA, ...)
nuclear DNA epigenetic changes	stable, age-related methylation pattern changes, heterochromatin decondensation & retrotransposons	OA, T2D, AD, diabetic retinopathy
non-DNA mitochondria structural issues	cardiolipin peroxidation & cristae collapse, stably altered ER-Mitochondria Tethering (MAMs)?	sarcopenia, HF, AD

Aggregate pipeline

Much progress, but many holes (subproblems unaddressed yet) & many of these programs will fail.

[programs of aging focused biotechs only; not exhaustive]

We don't yet know how beneficial combinations of many of these will be.

company/program

phase: pre 1 2 3 approved

aging subpathology

subpathology subcategory

supercategories	stable structural subpathologies [lists not exhaustive]	subcategories [lists not exhaustive]	companies/programs [lists not exhaustive]	pre-clinical	phase 1	phase 2	phase 3	approved	indication
long-lived ("death resistant") pathological cells	(long-lived) senescent cells	many							
		senescent keratinocytes	Rubedo's RLS-1496						Actinic Keratosis (AK)
	non-senescent stable pathological cells	senescent fibroblasts	Rubedo's RLS-1496						Actinic Keratosis (AK)
		senescent preadipocytes	Deciduosa via iNKT cells (in lung)						
		SnC endothelial cells & others	Deciduosa via iNKT cells						
		many	Arda Therapeutics						
		anergic T-cells & others							
	extracellular matrix remodeling & stiffening	AGEs & collagen crosslinks elastin fragmentation lens crystallin crosslinking vascular calcification	Revel's enzyme platform Elastin's elastin-mAb-LNPs Lento Bio						
	protein misfolding & aggregates	many	Attralus's platform						
		ATTR	Origami Therapeutics platform						ATTR-CM
		AL	several						Systemic AL (light chain) amyloidosis
		p-synuclein, tau, Aβ, others	Attralus's AT-02						
	intracellular toxic molecules	7-ketcholesterol	Cyclarity's UDP-003						Atherosclerosis
		unesterified free cholesterol	Cyclo Tx's Trappsol Cyclo						Niemann-Pick type C
		lipofuscin	Repair Bio's CDP platform						Familial Hypercholesterolemia
likely intimately related: stem cell degradation likely partially or largely epigenetic & epigenetic cell rejuvenation likely helps rescue stem cell degradation and cell loss / tissue atrophy	cell loss / tissue atrophy & stem cell degradation	neural SC depletion & demyelination	Renewal Bio platform						ALS
			ImmuneBridge cell eng platform						PD
			Brainstorm's NurOwn						PD
			Blue Rock's BRT-DA01						PD
			Aspen Neuroscience's iPSCs						Epilepsy
			Algorae's NTCELL						Spinal cord injury
			Neurona's NRTX-1001						AD
			Lineage Cell Tx's iPSCs						MS
			NeuroNascent's NNI-362						HF
			FibroBiologics' CYMS101						knee OA, sarcopenic obesity
	muscle loss / SC degradation		Mesoblast's rexlimestrocel-L						Age-related frailty, HLHS
			Immunis's IMM01-STEM						Age-related muscle decline
			Longeveron's Lomecel-B						Female stress urinary incontinence
			Unlimited Bio						Myotonic dystrophy type 1 (DM1)
			Muvon Therapeutics lead						Musculoskeletal defects
	nuclear DNA epigenetic changes	stable methylation pattern changes	Juvena's JUV-161						NAION, Glaucoma
			Ossium's OssiGraft						Age-related hearing loss, Liver fibrosis
			Life Biosciences's ER-100						T2D
		heterochromatin decondensation & retrotransposons	Shift Bio's platform						
			Junevity's platform						
			NewLimit's platform						
	nuclear DNA changes	many	Matter Bio						Metastatic PDAC
		telomere shortening	Elorigen's ZSCAN4						Telomere disorders, Bone marrow Failure
			Telocyte						AD
		non-mal. clonal expansion, inc CHIP cancer	Rejuvenation Technologies						IPF, Chronic Liver Disease
			[out of scope as an entirely separate biotechnology field]						
	mitochondria issues	many	Minovia's MNV-201						Pearson's Syndrome
			Cellegy's mito replacement plat.						
			Celvie's mito replacement plat.						
		4977-bp mtDNA deletion	Luca's mito replacement platform						
		mitochondrial clonal expansion	Mitrix's mito replacement platform						
		other misc. mutations							
			Gensight Biologics GS010						LHON

[as of spring 2026]

avg (min) years since founding to get to stage:

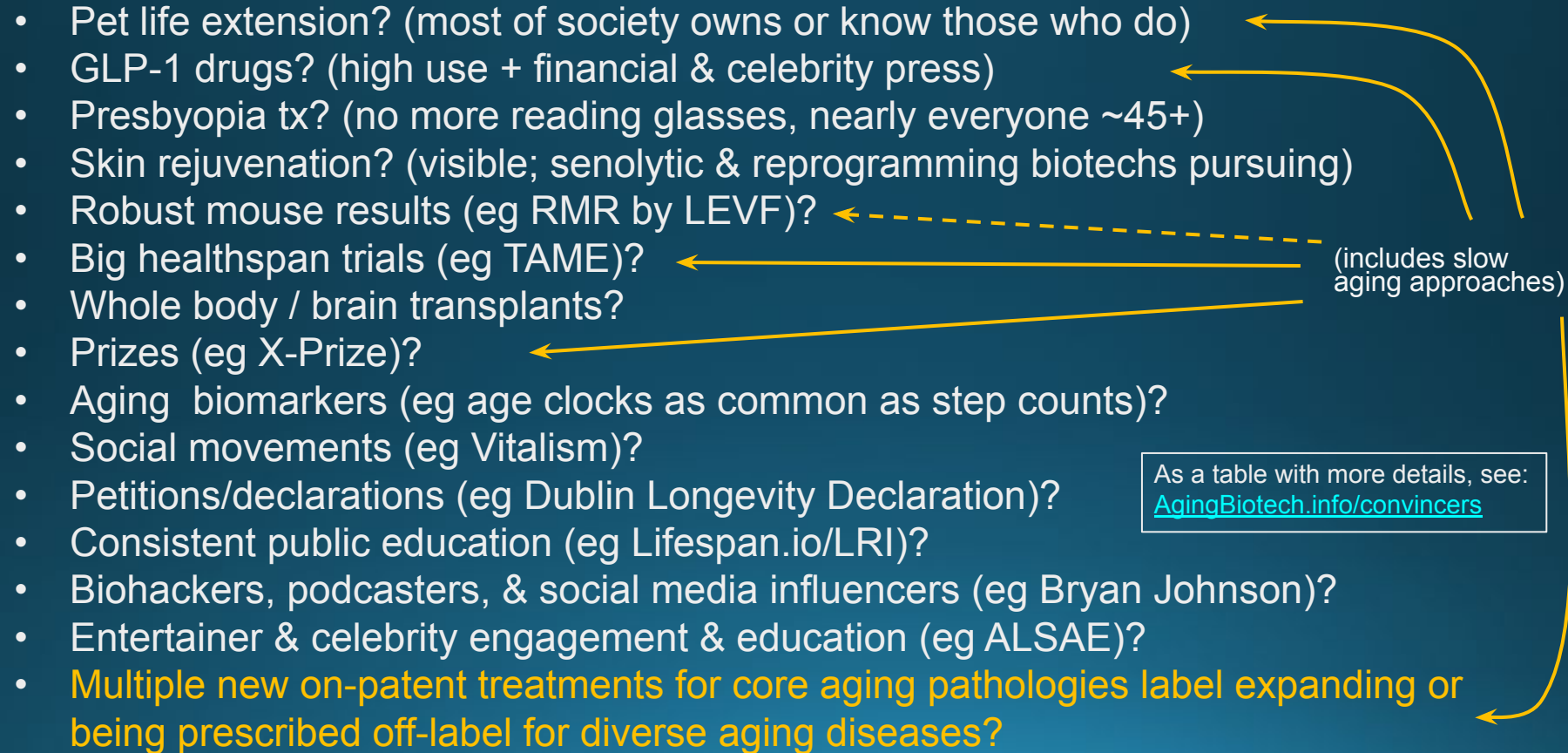
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Aggregate pipeline: my portfolio

phase: pre 1 2 3 approved

supercategories	stable structural subpathologies (lists not exhaustive)	subcategories (lists not exhaustive)	companies/programs (lists not exhaustive)	pre-clinical	phase 1	phase 2	phase 3	approved	indication
long-lived ("death resistant") pathological cells	(long-lived) senescent cells	many	Oisin's platform						Actinic Keratosis (AK) Actinic Keratosis (AK)
		senescent keratinocytes	Rubedo's RLS-1496						
		senescent fibroblasts	Rubedo's RLS-1496						
		senescent preadipocytes	Deciduous via iNKT cells (in lung)						
	non-senescent stable pathological cells	SnC endothelial cells & others	Deciduous via iNKT cells						
		many	Arda Therapeutics						
		anergic T-cells & others							
	extracellular matrix remodeling & stiffening	AGEs & collagen crosslinks	Revel's enzyme platform						
		elastin fragmentation	Elastin's elastin-mAb-LNPs						
		lens crystallin crosslinking	Lento Bio						
		vascular calcification							
	protein misfolding & aggregates	many	Attralus's platform						ATTR-CM Systemic AL (light chain) amyloidosis
		ATTR	Origami Therapeutics platform						
		AL	several						
		α -synuclein, tau, A β , others	Attralus's AT-02						
	intracellular toxic molecules	7-ketocholesterol	Cyclicity's UDP-003						Atherosclerosis
		unesterified free cholesterol	Cyclo Tx's Trappsol Cyclo						Niemann-Pick type C
		lipofuscin	Repair Bio's CDP platform						Familial Hypercholesterolemia
likely intimately related: stem cell degradation likely partially or largely epigenetic & epigenetic cell rejuvenation likely helps rescue stem cell degradation and cell loss / tissue atrophy	cell loss / tissue atrophy & stem cell degradation	many	Renewal Bio platform						ALS PD PD PD Epilepsy Spinal cord injury AD MS HF knee OA, sarcopenic obesity Age-related frailty, HLHS Age-related muscle decline Female stress urinary incontinence Myotonic dystrophy type 1 (DM1) Musculoskeletal defects
			ImmuneBridge cell eng platform						
			Brainstorm's NurOwn						
			Blue Rock's BRT-DA01						
			Aspen Neuroscience's iPSCs						
			Algorae's NTCELL						
			Neurona's NRTX-1001						
			Lineage Cell Tx's iPCSs						
			NeuroNascent's NNI-362						
			FibroBiologics' CYMS101						
	muscle loss / SC degradation		Mesoblast's rexiemestrocel-L						
			Immunis's IMM01-STEM						
			Longeveron's Lomecel-B						
			Unlimited Bio						
			Muvon Therapeutics lead						
			Juvena's JUV-161						
			Ossium's OssiGraft						
	nuclear DNA epigenetic changes	stable methylation pattern changes	Life Biosciences's ER-100						NAION, Glaucoma
			Shift Bio's platform						Age-related hearing loss, Liver fibrosis
			Juvenity's platform						T2D
			NewLimit's platform						
		heterochromatin decondensation & retrotransposons							
	nuclear DNA changes	many	Matter Bio						Metastatic PDAC
		telomere shortening	Elixirgen's ZSCAN4						Telomere disorders, Bone marrow Failure
		non-mal. clonal expansion, inc CHIP cancer	Telocyte						AD
			Rejuvenation Technologies						IPF, Chronic Liver Disease
			[out of scope as an entirely separate biotechnology field]						
	mitochondria issues		Minovia's MNV-201						Pearson's Syndrome
			Cellergy's mito replacement plat.						
			Cellvie's mito replacement plat.						
			Luca's mito replacement platform						
		4977-bp mtDNA deletion	Mitrix's mito replacement platform						
		mitochondria clonal expansion							
		other misc. mutations	Gensight Biologics GS010						LHON
[as of spring 2026]				avg (min) years since founding to get to stage:	12.5 (7)	22 (10)			

Anything is on the path if it helps trigger a sea change of public awareness, support, & funding, but which will help?

- Pet life extension? (most of society owns or know those who do)
 - GLP-1 drugs? (high use + financial & celebrity press)
 - Presbyopia tx? (no more reading glasses, nearly everyone ~45+)
 - Skin rejuvenation? (visible; senolytic & reprogramming biotechs pursuing)
 - Robust mouse results (eg RMR by LEVF)?
 - Big healthspan trials (eg TAME)?
 - Whole body / brain transplants?
 - Prizes (eg X-Prize)?
 - Aging biomarkers (eg age clocks as common as step counts)?
 - Social movements (eg Vitalism)?
 - Petitions/declarations (eg Dublin Longevity Declaration)?
 - Consistent public education (eg Lifespan.io/LRI)?
 - Biohackers, podcasters, & social media influencers (eg Bryan Johnson)?
 - Entertainer & celebrity engagement & education (eg ALSAE)?
 - **Multiple new on-patent treatments for core aging pathologies label expanding or being prescribed off-label for diverse aging diseases?**
- (includes slow aging approaches)
- As a table with more details, see: AgingBiotech.info/convincers
- 

Broadly slowing aging vs. divide-and-conquer rejuvenation: tradeoffs

- Industrialized societies suffer high prevalence of lifestyle-mediated age acceleration (from poor diet, inactivity, sleep deprivation...; & resulting obesity)
- Lifestyle makes a 10-20 year difference in human lifespan ([sources & analysis](#))
- Broadly slowing aging has breadth so yields immediate effects on healthspan, especially by mitigating the lifestyle-based age acceleration (most pharma \$ drawn to this!)
- But existing data suggest that **the tradeoff for slow aging approaches having benefits against most aging subpathologies is not coming close to totally eliminating any** & having limited additive effects, so limited long-term total effect sizes (eg, <50% healthspan/lifespan extension in mice, small % in humans)
- **Divide-and-conquer rejuvenation** is the default path to fully eliminating aging pathology in the long run, and it **can deliver full elimination of some subpathologies in the short-term**, with beneficial effects vs. multiple diseases
- With subpathology-specific biomarkers, trials need lower n to show efficacy
- Individual therapies don't broadly rescue common lifestyle age acceleration (but a program of many will, & go further esp. as it approaches comprehensiveness)

Incentives lacking for combination work

To my knowledge **this is the first ever broad assessment of the clinical translational status of the divide-and-conquer approach to rejuvenation** or fully eliminating aging (such assessment has only become possible in the past ~5yrs).

Prior SENS Research Foundation status summaries from many years ago were from a time before many companies had even spun out to directly address stable structural aging subpathologies & from when all were still far from starting clinical trials.

A lot of this you won't hear in most discussion of the aging/longevity field because there's no little incentive in academia or biotech to combine the work of others. So right now, combining therapies is left to only the very last step: clinical medical practice, with no systematic study of how total effectiveness scales with # of therapies or category.

Poll:

What would you like to hear more about in Q&A?

These slides: <https://tinyurl.com/AgingPipeline26> or QR:



Take-homes to remember

- Before the mid-1800s, eliminating top killer infectious diseases seemed fanciful
- Therapies targeting core aging areas are making it to phase 3 and to approvals
- Aging research receives 80x less US government funding per death than cancer
- Broadly slowing aging & divide-&-conquer rejuvenation are different subfields
- Divide-&-conquer progress is happening but must be judged by effective coverage (not 1-intervention lifespan)
- We don't yet know how good aging therapies will be in combination

Questions?

These slides: <https://tinyurl.com/AgingPipeline26> or QR:





"I also treat my body like a temple: an ancient structure in desperate need of repair, long ignored and of rapidly decreasing significance to a largely indifferent society,"

Aggregate pipeline:

(squished from a traditional bar chart into a table)

template, i.e. scope of the problem (at least, not exhaustive)

stable structural aging subpathologies ↓							
supercategories	main categories	specific subcategories & company programs addressing this area therapeutically					
long-lived ("death resistant") pathological cells	(long-lived) senescent cells	senescent fibroblasts	senescent keratinocytes	senescent endothelial cells	senescent preadipocytes	senescent immune cells	
	non-senescent stable pathological cells						
		exhausted/anergic T-cells	persistent myofibroblasts	hypertrophic/inflamed adipocytes	macrophage foam cells (see intracell toxins / cholesterol)	CHIP (see DNA / clonal expansion)	
	extracellular matrix remodeling & stiffening	AGEs & collagen crosslinking	elastin fragmentation	vascular calcification	lens crystallin crosslinking		
	protein misfolding & aggregates	ATTR	α-synuclein	tau	Aβ	TDP-43	AL
	intracellular toxic molecules	lipofuscin	7-ketocholesterol	unesterified free cholesterol			
likely intimately related: stem cell degradation likely partially or largely epigenetic & epigenetic cell rejuvenation likely helps rescue stem cell degradation and cell loss / tissue atrophy	cell loss / tissue atrophy & stem cell degradation	neural stem cell depletion & neuron demyelination	muscle satellite cell degradation / muscle loss	thymus involution	cartilage loss	vascular network degradation	misc. other cell loss/degradation
	nuclear DNA epigenetic changes	stable methylation pattern changes	heterochromatin decondensation & retrotransposons				
	nuclear DNA changes	telomere shortening	non-malignant clonal expansion (inc. CHIP)	cancer mutations [out of scope as entirely separate field]			
structural mitochondrial aging	mitochondria DNA mutations	4977-bp mtDNA deletion	mitochondria clonal expansion	other mutations			
	non-DNA mitochondria structural issues	cardiolipin peroxidation & cristae collapse	stably altered ER-Mitochondria Tethering?				
	barrier integrity degradation	BBB	gut	skin?			
color key →	not yet but maybe in the works (spinning out from academia)	company failed financially or pivoted away but approach still scientifically valid	pre-clinical	ph.1	ph.2	ph.3	approved

Aggregate pipeline:

my investment portfolio

We need more investors focused on this, especially attacking it comprehensively.

supercategories	main categories	specific subcategories & company programs addressing this area therapeutically					
long-lived ("death resistant") pathological cells	(long-lived) senescent cells	Oislin's platform for all SnCs based on overexpression of p16 (or p21 or p53)					
		senescent fibroblasts	senescent keratinocytes	senescent endothelial cells	senescent preadipocytes	senescent immune cells	
		Rubedo's RLS-1496	Rubedo's RLS-1496				
	non-senescent stable pathological cells	Deciduous via iNKT cells (in lung)			Deciduous via iNKT cells		
		exhausted/anergic T-cells	persistent myofibroblasts	hypertrophic/inflamed adipocytes	macrophage foam cells [see intracell toxins / cholesterol]	CHIP [see DNA / clonal expansion]	
	extracellular matrix remodeling & stiffening	AGEs & collagen crosslinking Revel Pharma's enzyme engineering platform	elastin fragmentation Elastrin's elastin-mAb-LNPs	vascular calcification	lens crystallin crosslinking		
	protein misfolding & aggregates	Origami Tx's protein degraders platform					
		Endear Therapies' platform					
		Covalent Bio's catalytic antibodies					
		ATTR	α-synuclein	tau	Aβ	TDP-43	AL
	intracellular toxic molecules	lipofuscin	7-ketocholesterol Cyclarity's UDP-003	unesterified free cholesterol Repair Bio's cholesterol degrading platform			
		stealth reprogramming company ImmuneBridge cell engineering platform stealth EV company					
	cell loss / tissue atrophy & stem cell degradation	neural stem cell depletion & neuron demyelination Renu Bio's lead program stealth glial replacement company	muscle satellite cell degradation / muscle loss Immunis's IMM01-STEM Juvena's JUV-161	thymus involution Lygenesis thymus program (indirect portco) not a done deal but newco about to spin out	cartilage loss not a done deal but newco about to spin out	vascular network degradation	misc. other cell loss/degradation
	nuclear DNA epigenetic changes	stable methylation pattern changes [below here better at the supercategory level?] Shift Bio's platform Junevity's platform Turn Bios' OSKMLN platform	heterochromatin decondensation & retrotransposons				
	nuclear DNA changes	telomere shortening Rejuvenation Technologies	non-malignant clonal expansion (inc. CHIP)	cancer mutations [out of scope as entirely separate field]			
		Cellergy's mitochondria replacement platform					
	structural mitochondrial aging	mitochondria DNA mutations	4977-bp mtDNA deletion	mitochondria clonal expansion	other mutations		
		non-DNA mitochondria structural issues	cardiolipin peroxidation & cristae collapse Retrotape D-PUFAs	stably altered ER-Mitochondria Tethering?			
	barrier integrity degradation	BBB Reservoir Neuro's platform	gut	skin?			
	color key →	not yet but maybe in the works (spinning out from academia)	company failed financially or pivoted away but approach still scientifically valid	pre-clinical	ph.1	ph.2	ph.3 approved

Aggregate pipeline:

aging focused biotech, phase 1 on

(no veterinary or supplements, misses programs in companies not aging focused, but some of these will fail)

supercategories	main categories	specific subcategories & company programs addressing this area therapeutically						
long-lived ("death resistant") pathological cells	(long-lived) senescent cells	senescent fibroblasts Rubedo's RLS-1496	senescent keratinocytes Rubedo's RLS-1496	senescent endothelial cells	senescent preadipocytes	senescent immune cells		
	non-senescent stable pathological cells	exhausted/anergic T-cells	persistent myofibroblasts	hypertrophic/inflamed adipocytes	macrophage foam cells [see intracell toxins / cholesterol]	CHIP [see DNA / clonal expansion]		
	extracellular matrix remodeling & stiffening	AGEs & collagen crosslinking	elastin fragmentation	vascular calcification	lens crystallin crosslinking			
	protein misfolding & aggregates	ATTR [many approvals slow progression without being rejuvenative, but maybe slow by a lot]	α-synuclein	tau	Aβ	TDP-43	AL	Attralus's AT-02
	intracellular toxic molecules	lipofuscin	7-ketocholesterol Cyclarity's UDP-003	unesterified free cholesterol Cyclo Therapeutics's Trappsol Cyclo				
likely intimately related: stem cell degradation likely partially or largely epigenetic & epigenetic cell rejuvenation likely helps rescue stem cell degradation and cell loss / tissue atrophy	cell loss / tissue atrophy & stem cell degradation	neural stem cell depletion & neuron demyelination Brainstorm's NurOwn Blue Rock's BRT-DA01 Aspen Neuroscience's iPSCs Algorae's NTCELL Neurona's NRTX-1001 Lineage Cel Tx's iPSCs NeuroNascent's NNI-362 FibroBiologics' CYMS101	muscle satellite cell degradation / muscle loss Mesoblast's rexiemestrocet-L Immunis's IMM01-STEM Longeveron's Lomemel-B Muvon Therapeutics lead Juvena's JUV-161 Ossium's Ossigraft (section 361 commercial)	thymus involution Intervene Immune's TRIIM	cartilage loss Medipost Caristem (Korea, ph3 Japan?, US) Mesoblast's rexiemestrocet-L Cynata' CYP-004 Pluri's PLX-PAD	vascular network degradation Athersys's MultiStem Mesoblast's rexiemestrocet-L Hemostemix's ACP-01	misc. other cell loss/degradation Endogene's EA-2353 (eye) Lineage Cell's iPSCs (eye)	
	nuclear DNA epigenetic changes	stable methylation pattern changes Life Biosciences's ER-100	heterochromatin decondensation & retrotransposons					
	nuclear DNA changes	telomere shortening Elixirgen's ZSCAN4	non-malignant clonal expansion (inc. CHIP)	cancer mutations [out of scope as entirely separate field]				
structural mitochondrial aging	mitochondria DNA mutations	Minovia's MNV-201						
	non-DNA mitochondria structural issues	4977-bp mtDNA deletion	mitochondria clonal expansion	other mutations Gensight Biologics GS010				
	barrier integrity degradation	BBB	gut	skin?				
color key →	not yet but maybe in the works (spinning out from academia)	company failed financially or pivoted away but approach still scientifically valid	pre-clinical	ph.1	ph.2 (years since founding: min 7, median 12.5)	ph.3 (yrs f/ founding: min 10, med 22)	approved	

Aggregate pipeline:

aging focused biotech, inc. preclin

Not exhaustive, problems or therapies.

We have no idea how beneficial combinations of many of these will be.

supercategories	main categories		specific subcategories & company programs addressing this area therapeutically				
long-lived ("death resistant") pathological cells	(long-lived) senescent cells	Oisin's platform for all SnCs based on overexpression of p16 (or p21 or p53)					
		senescent fibroblasts	senescent keratinocytes	senescent endothelial cells	senescent preadipocytes	senescent immune cells	
		Rubedo's RLS-1496	Rubedo's RLS-1496		Deciduous via iNKT cells		
		Deciduous via iNKT cells (in lung)					
	non-senescent stable pathological cells	Arda Therapeutics					
		exhausted/anergic T-cells	persistent myofibroblasts	hypertrophic/inflamed adipocytes	macrophage foam cells	CHIP [see DNA / clonal expansion]	
					[see intracell toxins / cholesterol]		
	extracellular matrix remodeling & stiffening	AGEs & collagen crosslinking	elastin fragmentation	vascular calcification	lens crystallin crosslinking		
		Revel Pharma's enzyme engineering platform	Elastin's elastin-mAb-LNPs		Lento Bio		
	protein misfolding & aggregates	Atralus's platform					
		Origami Therapeutics platform					
		ATTR	α-synuclein	tau	Aβ	TDP-43	AL
		[many approvals slow progression without being rejuvenative, but maybe slow by a lot]					Atralus's AT-02
	intracellular toxic molecules	lipofuscin	7-ketocholesterol	unesterified free cholesterol			
			Cyclarity's UDP-003	Cyclo Therapeutics's Trappsol Cyclo			
				Repair Bio's cholesterol degrading			
likely intimately related: stem cell degradation likely partially or largely epigenetic & epigenetic cell rejuvenation likely helps rescue stem cell degradation and cell loss / tissue atrophy	cell loss / tissue atrophy & stem cell degradation	Renewal Bio platform					
		ImmuneBridge cell engineering platform					
		neural stem cell depletion & neuron demyelination	muscle satellite cell degradation / muscle loss	thymus involution	cartilage loss	vascular network degradation	misc. other cell loss/degradation
		Brainstorm's NurOwn	Mesoblast's rexlemestrocel-L	Intervene Immune's TRIIM	Medipost Cartistem (Korea, ph3 Japan, US)	Athersys's MultiStem	Endogene's EA-2353 (eye)
		Blue Rock's BRT-DA01	Immunis's IMM01-STEM	Thymune Therapeutics	Mesoblast's rexlemestrocel-L	Mesoblast's rexlemestrocel-L	Lineage Cell's iPCs (eye)
		Aspen Neuroscience's iPSCs	Longeveron's Lomecel-B	Tolerance Bio	Cynata' CYP-004	Hemostemix's ACP-01	
		Algorae's NTECELL	Unlimited Bio	TECregen	Plun's PLX-PAD	Unlimited Bio	
		Neurona's NRTX-1001	Muvon Therapeutics lead				
		Lineage Cel Tx's iPCs	Juvena's JUV-161				
		NeuroNascent's NNI-362	Ossium's Ossigraft (section 361 commercial)				
	FibroBiologics' CYMS101						
	nuclear DNA epigenetic changes	stable methylation pattern changes [below here better at the supercategory level?]	heterochromatin decondensation & retrotransposons				
		Life Biosciences's ER-100					
		Shift Bio's platform					
		Junevity's platform					
	NewLimit's platform						
	nuclear DNA changes	Matter Bio					
		telomere shortening	non-malignant clonal expansion (inc. CHIP)	cancer mutations			
		Elxirgen's ZSCAN4		[out of scope as entirely separate field]			
		Telocyte					
		Rejuvenation Technologies					
structural mitochondrial aging		Minovia's MNV-201					
		Cellergy's mitochondria replacement platform					
		Cellvie's mitochondria replacement platform					
		Luca's mitochondria replacement platform					
		Mitrix's mitochondria replacement platform					
	mitochondria DNA mutations	4977-bp mtDNA deletion	mitochondria clonal expansion	other mutations			
				Gensight Biologics GS010			
	non-DNA mitochondria structural issues	cardiolipin peroxidation & cristae collapse	stably altered ER-Mitochondria Tethering?				
	barrier integrity degradation	BBB	gut	skin?			

Aggregate pipeline:

diagnostics
(poor coverage)

stable structural aging subpathologies ↓	supercategories	main categories	specific subcategories & clinical diagnostics					
long-lived ("death resistant") pathological cells	(long-lived) senescent cells	senescent fibroblasts	senescent keratinocytes	senescent endothelial cells	senescent preadipocytes	senescent immune cells		
		non-senescent stable pathological cells	exhausted/anergic T-cells	persistent myofibroblasts	hypertrophic/inflamed adipocytes	macrophage foam cells (see intracell toxins / cholesterol)	CHIP (see DNA / clonal expansion)	
	extracellular matrix remodeling & stiffening	AGEs & collagen crosslinking	elastin fragmentation	vascular calcification	lens crystallin crosslinking			
				CCTA				
	protein misfolding & aggregates	Amprion platform						
		ATTR	α-synuclein	tau	Aβ	TDP-43	AL	
		scan (non-clinical)	Amprion	some scans & related blood tests, but clinical use uneven				
	intracellular toxic molecules	lipofuscin	7-ketocholesterol	unesterified free cholesterol				
likely intimately related: stem cell degradation likely partially or largely epigenetic & epigenetic cell rejuvenation likely helps rescue stem cell degradation and cell loss / tissue atrophy	cell loss / tissue atrophy & stem cell degradation	neural stem cell depletion & neuron demyelination	muscle satellite cell degradation / muscle loss	thymus involution	cartilage loss	vascular network degradation	misc. other cell loss/degradation	
			DEXA indirectly, functional tests	scans (non-clinical?)	MRI (only individually, joint by joint)		DEXA (bone)	
	nuclear DNA epigenetic changes	stable methylation pattern changes	heterochromatin decondensation & retrotransposons					
	nuclear DNA changes	telomere shortening	non-malignant clonal expansion (inc. CHIP)	cancer mutations				
		mostly only for blood & not critically short only		[out of scope as entirely separate field]				
structural mitochondrial aging	mitochondria DNA mutations	4977-bp mtDNA deletion	mitochondria clonal expansion	other mutations				
	non-DNA mitochondria structural issues	cardiolipin peroxidation & cristae collapse	stably altered ER-Mitochondria Tethering?					
	barrier integrity degradation	BBB	gut	skin?				
color key →	not yet but maybe in the works (spinning out from academia)	company failed financially or pivoted away but approach still scientifically valid	pre-commercial	commercial				

Additional dimensions for the 'divide' of
divide-and-conquer...

The whole body: compartment/organ & tissue-type/cell-type

[illegible]

Species: humans, mice, dogs

mice:

- LEVF trying combos
- no one is capturing biotech preclinical therapy data nor are the research-grade treatments companies create on the way to IND broadly usable by others

dogs:

- now a handful of companies not just 1
- but most using broad slow-aging approaches
- not enough stable structural rejuvenation therapies yet for dogs for meaningful aggregate pipeline

Ironically, things with small effect size (obesity, metabolism) attract huge \$\$\$ distorting company incentives & programs

Example companies (just in my portfolio) where focus/programs altered by the huge gravitational pull of money in obesity & metabolism:

- Immunis
- Juvena
- Oisin
- Junevity
- Gordian
- (also Bioage—not in my portfolio but almost was & its founding was the inspiration for my own investing start)

Many more examples outside my portfolio.

Not so much a criticism of these companies—great if they can capture some of this money for the overall field, but still a lament of the phenomenon's consequence of drawing resources away from the other main subfield.