Institut für Finanz- und Aktuarwissenschaften

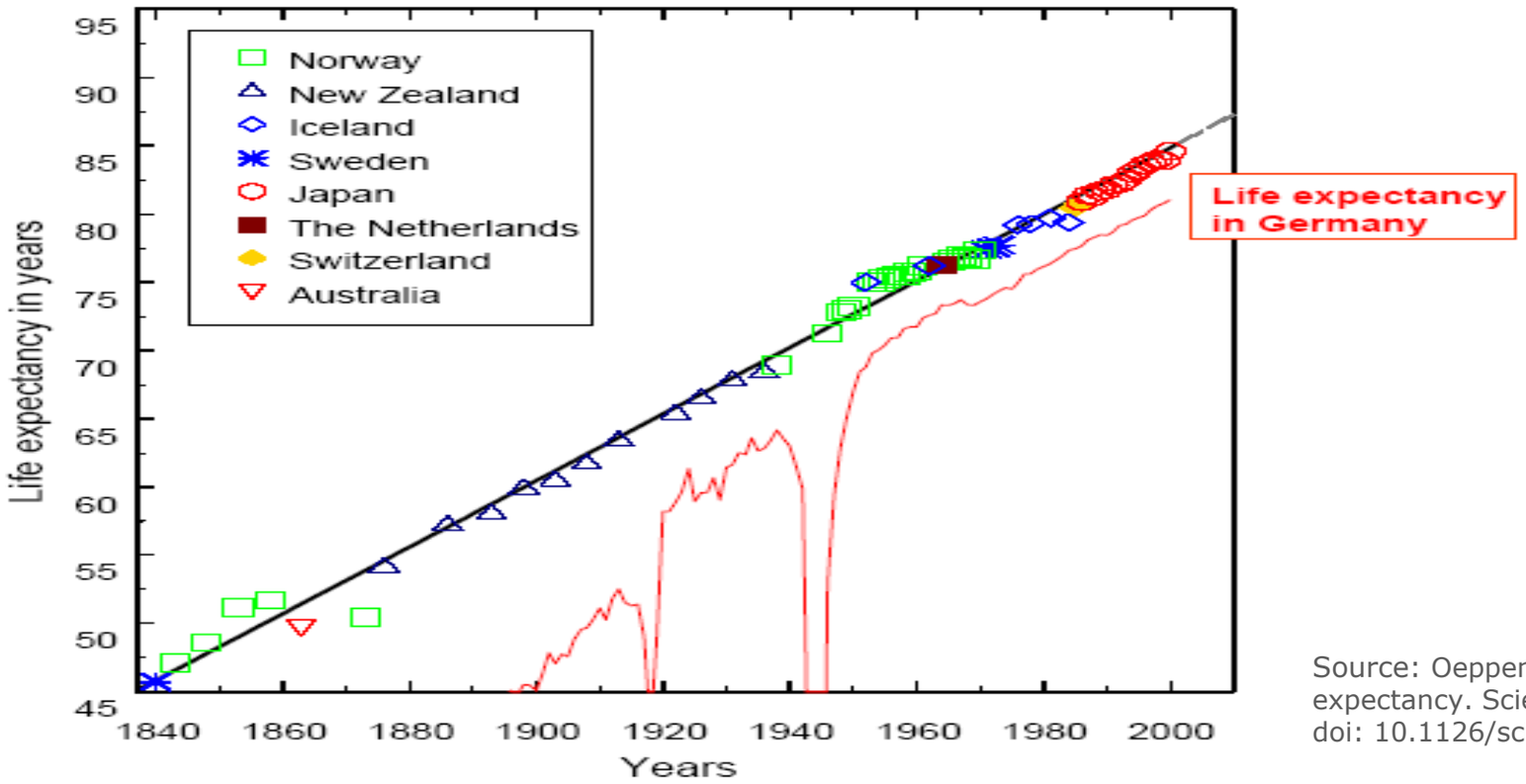
The future of human life expectancy

Do we know how little we know?

- Jochen Ruß

- November 2023

A well-known picture to start with



Source: Oeppen J, Vaupel JW. Demography. Broken limits to life expectancy. Science. 2002 May 10;296(5570):1029-31. doi: 10.1126/science.1069675. PMID: 12004104.

The so-called record life expectancy (life expectancy in the "healthiest" country in the world) has been rising linearly for a long time. Speed: approx. 2.5 years per decade

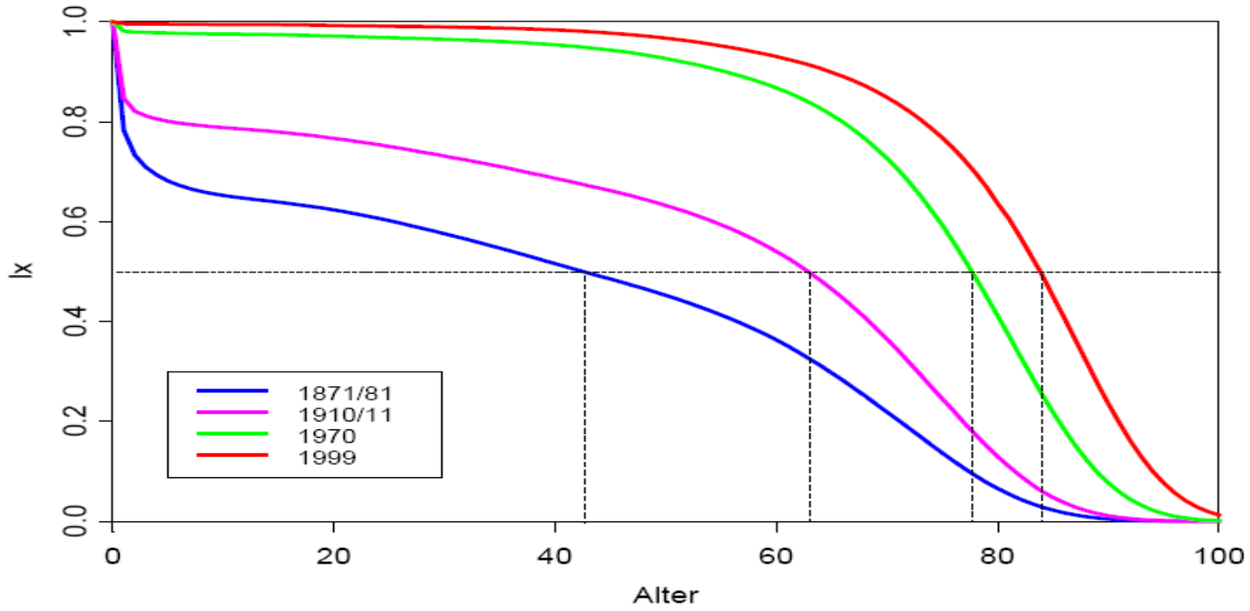
Will this go on forever?

Part 1: The Party is over!

There are numerous very good arguments to suggest that the increase in life expectancy will have to slow down significantly in the near future. Even a decline in life expectancy seems possible.

Arguments for a trend reversal

Argument 1: There has never been a "mechanism" that has uniformly increased life expectancy. Different things have influenced the "lx-curve" at different times in different ways. This has by pure chance led to a linear increase of LE (which is an aggregated parameter of this curve).

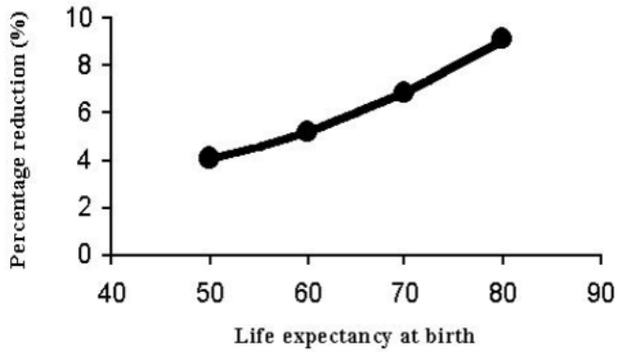


If the linear trend has emerged only by chance through a combination of a wide variety of independent effects, there is little to suggest that it will continue.

Arguments for a trend reversal

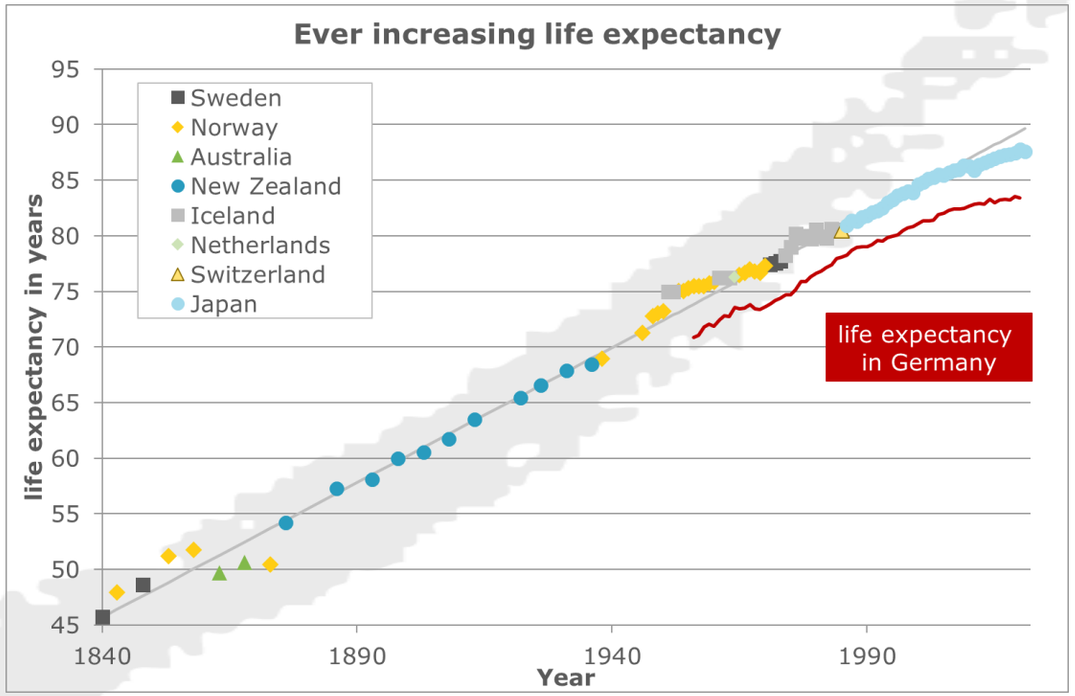
Argument 2: The relative reduction in mortality required to gain 1 year of additional LE is the larger, the larger the LE already is.

Percentage reduction in death rates at all ages required to raise life expectancy at birth by one year



SOURCE: Olshansky, Carnes and Désesquelles, 2001. Prospects for Human Longevity. *Science*.

Argument 3: The slowdown of the increase has already started.



Source: Until 1999: figures from the chart by Oeppen und Vaupel (2002) (see above); own calculations for the years afterwards.

In some countries (e.g., the USA), life expectancy is already decllining.

Arguments for a trend reversal

Argument 4: Numerous other risks that could potentially increase mortality.

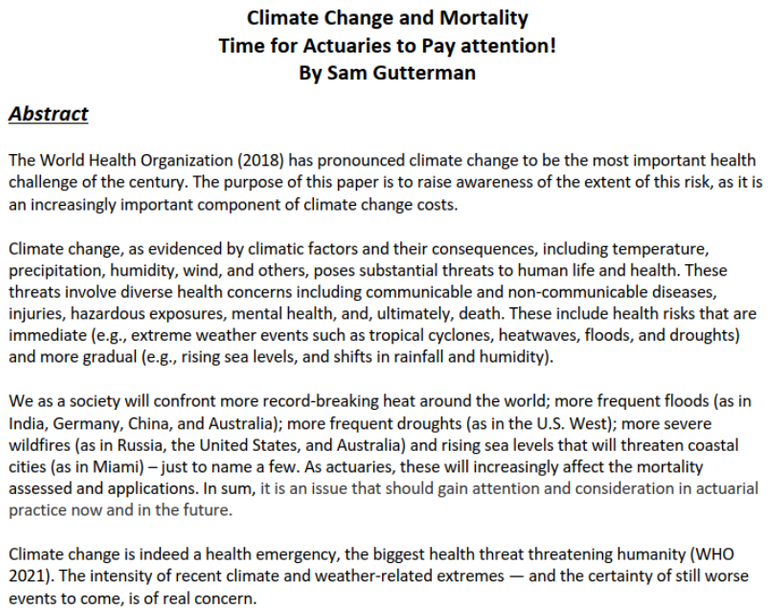
- Climate change
- Environment (microplastics, fine particulate matter,...)
- Long Covid
- Increased probability of future pandemics
- Lifestyle
- etc.

Remark:

- Anything caused by "behavioral factors" would lead to an increase in the heterogeneity of mortality in the population.
  - Annuitant mortality in insurers' portfolios likely to be less affected.
- Anything else could, in principle, affect the entire population.

Current paper on climate change

- it lists a wide range of possible effects



Climate Change and Mortality
Time for Actuaries to Pay attention!
By Sam Gutterman

Abstract

The World Health Organization (2018) has pronounced climate change to be the most important health challenge of the century. The purpose of this paper is to raise awareness of the extent of this risk, as it is an increasingly important component of climate change costs.

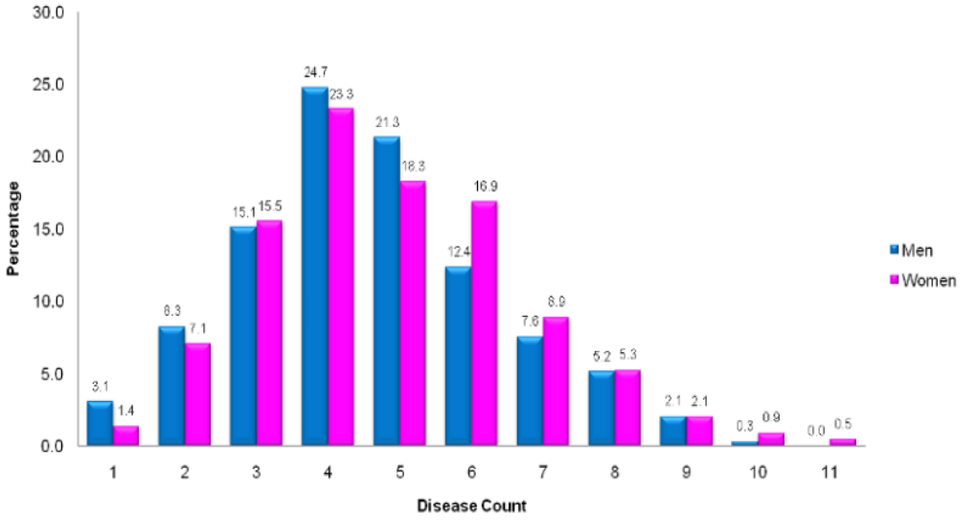
Climate change, as evidenced by climatic factors and their consequences, including temperature, precipitation, humidity, wind, and others, poses substantial threats to human life and health. These threats involve diverse health concerns including communicable and non-communicable diseases, injuries, hazardous exposures, mental health, and, ultimately, death. These include health risks that are immediate (e.g., extreme weather events such as tropical cyclones, heatwaves, floods, and droughts) and more gradual (e.g., rising sea levels, and shifts in rainfall and humidity).

We as a society will confront more record-breaking heat around the world; more frequent floods (as in India, Germany, China, and Australia); more frequent droughts (as in the U.S. West); more severe wildfires (as in Russia, the United States, and Australia) and rising sea levels that will threaten coastal cities (as in Miami) – just to name a few. As actuaries, these will increasingly affect the mortality assessed and applications. In sum, it is an issue that should gain attention and consideration in actuarial practice now and in the future.

Climate change is indeed a health emergency, the biggest health threat threatening humanity (WHO 2021). The intensity of recent climate and weather-related extremes — and the certainty of still worse events to come, is of real concern.

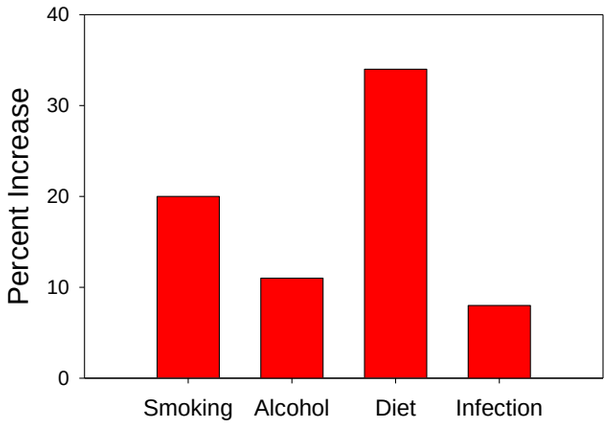
Arguments for a trend reversal

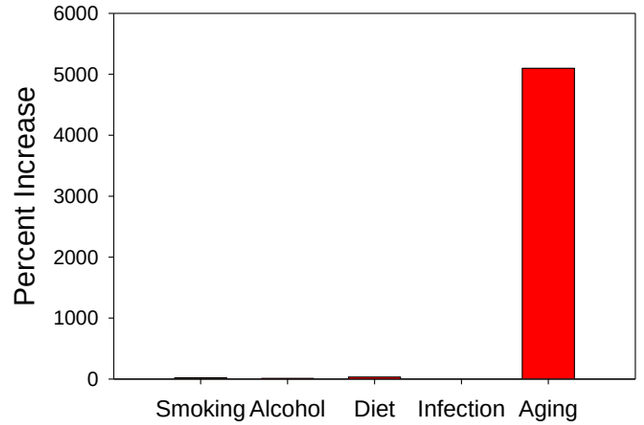
Argument 5: The higher the age, the less is to be gained from "typical medical progress". If you cure one disease in an 85-year-old, she will soon get (or already has) the next one (whack-a-mole effect).



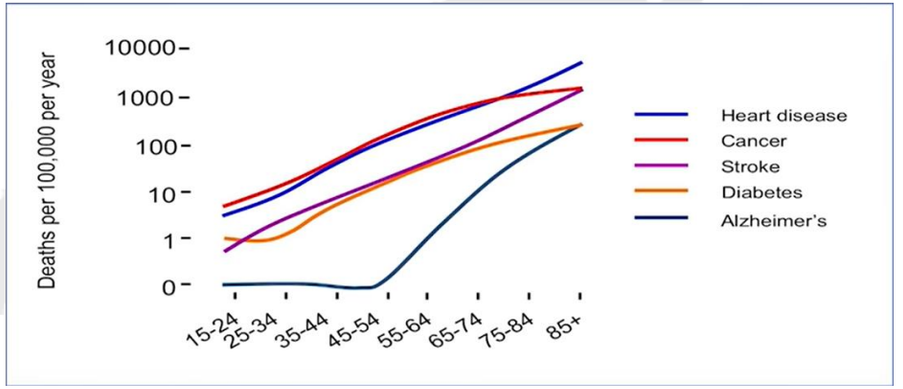
Source: Collerton et al (2009) British Medical Journal Health and disease in 85 year olds: baseline findings from the Newcastle 85+ cohort study

Conclusion: As long as it is not possible to slow down ageing per se, the end of the "longevity party" seems inevitable!





Source: S. Jay Olshansky (2016), presentation at the Longevity 12 Conference. The chart shows risk factors for cancer. The talk also includes images for heart disease and Alzheimer's disease.



Source: Nir Barzilai: How to die at a very old age. TEDx-Talk: https://www.youtube.com/watch?v=TsA4SHhUzt4

Part 2: The Party hasn't even started!

There are numerous very good arguments to suggest that the increase in life expectancy will accelerate significantly in the near future.

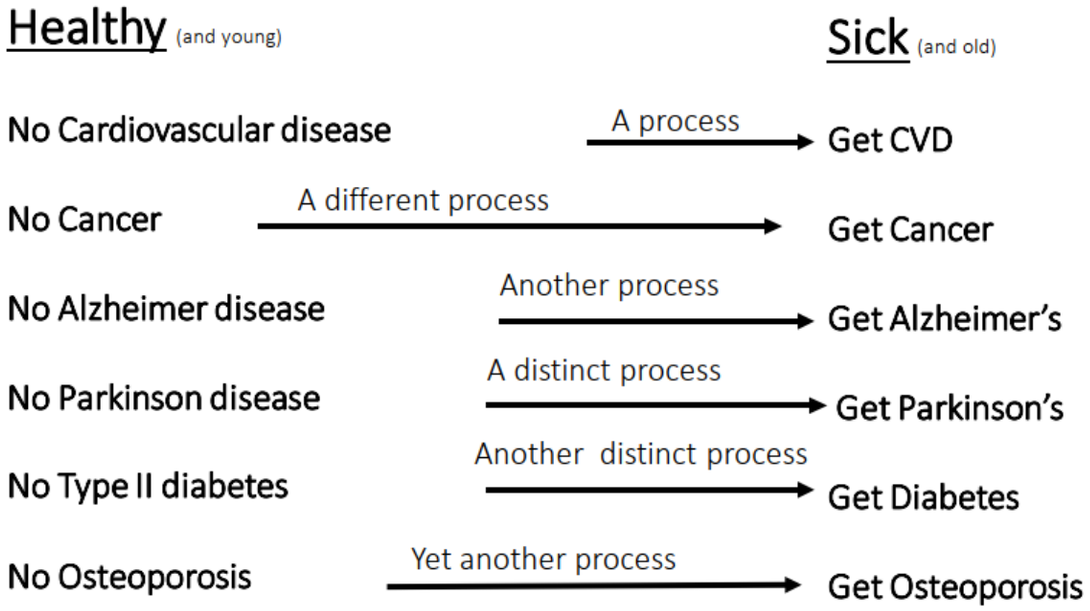
Being able to slow down ageing seems to be within reach.

In what follows, I present a subjective (!) selection of advances in this field.*

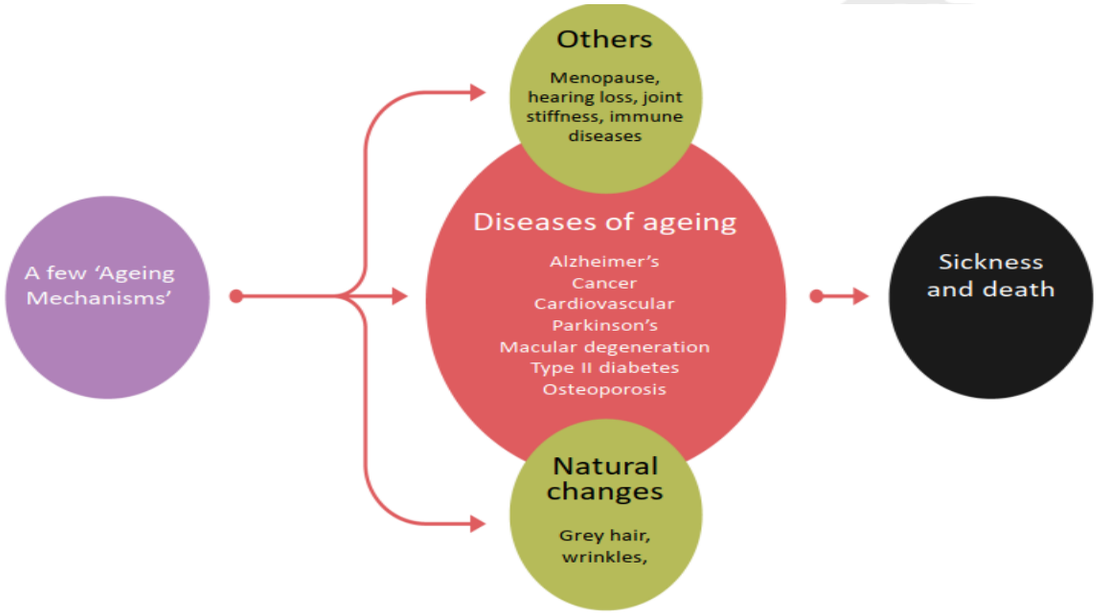
***Note**: There are numerous other developments that could have an impact on human LE by affecting individual diseases. Here also it is uncertain if and when progress will be made. However, because of the whack-a-mole effect, the potential impact is limited. Therefore, we do **not** address these aspects.
Disclaimer: This chapter deals largely with medical issues. The presenter is not an expert in this field. Therefore, no decisions should be made based on the following statements without independently verifying them.

The most important thing in advance: It is now understood what "ageing" actually is.

Ageing does not work like this…,



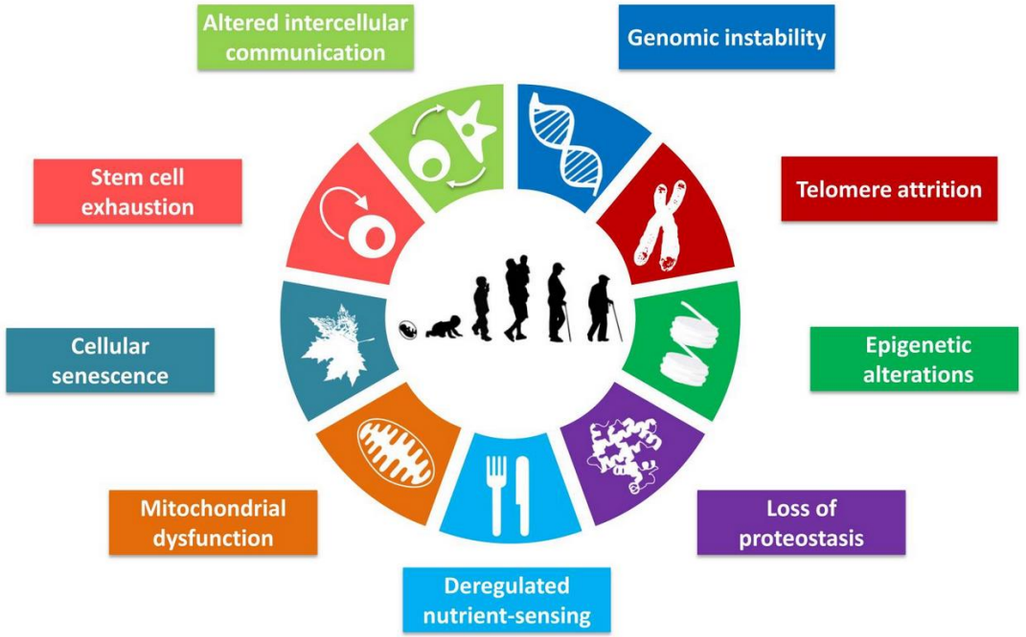
but rather like this:



Source: Biology of Human Senescence, presentation of Richard Faragher, Professor of Biogerontology, at the 2023 „Living to 100 Symposium".

What are the Ageing Mechanisms?

The most popular representation: Hallmarks of Ageing



An alternative approach - based on damage processes and "maintenance"

The "seven deadly things" & their fixes

Damage type	The maintenance approach
Cell loss, cell atrophy	Replace, using stem cells
Division-obsessed cells	Reinforce, using telomere control
Death-resistant cells	Remove, using suicide genes etc
Mitochondrial mutations	Reinforce, using backup copies
Intracellular waste products	Remove, using foreign enzymes
Extracellular waste products	Remove, using immune system
Extracellular matrix stiffening	Repair, using crosslink-breakers

Existence of any 8th is looking increasingly unlikely

Source (top): https://www.yourheights.com/blog/longevity/how-to-end-ageing-with-aubrey-de-grey/; Source (left): López-Otín, Carlos, et al. "The hallmarks of ageing." *Cell* 153.6 (2013): 1194-1217.

Consensus among experts seems to be: "Mechanisms that cause ageing are known and druggable!"*.

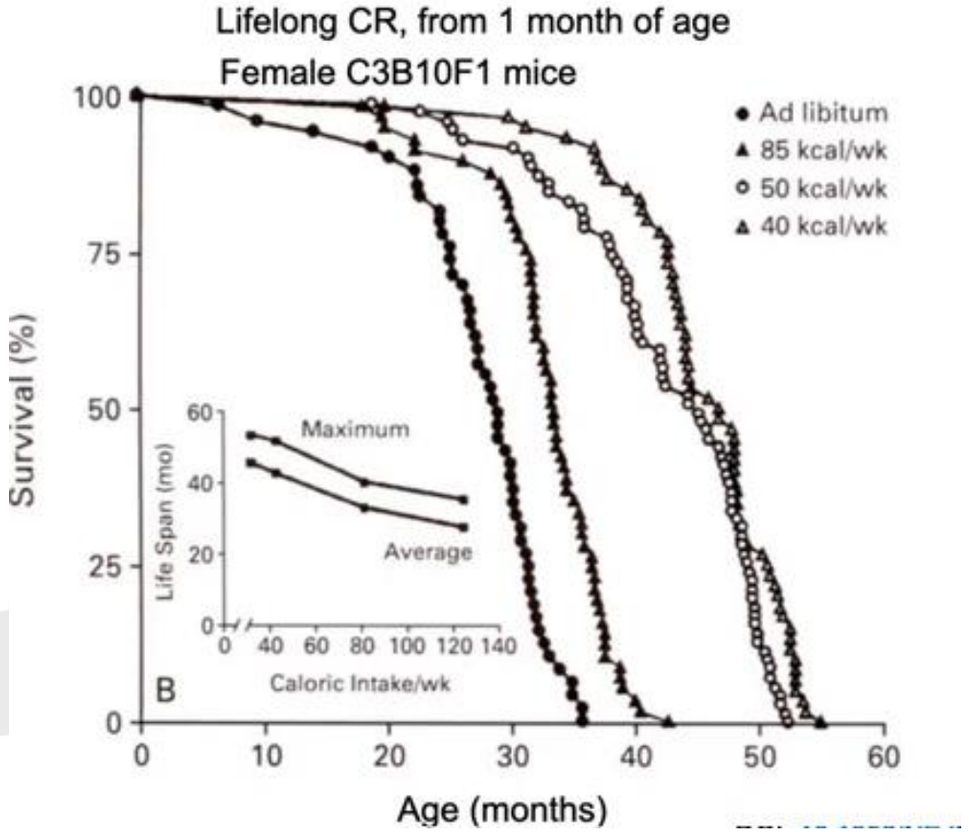
Geroscience is the science that targets the Hallmarks of Ageing.

* Quote is from the already cited talk „Biology of Human Senescence" by Richard Faragher.

Currently discussed approaches to influence the ageing process

Caloric Restriction (CR):

- Put simply: eat only enough to barely survive.
- This extends life expectancy in many species - in some significantly.
  - presumably also has a positive effect in humans
  - but who wants to do that permanently?
    - Therefore, hardly any long-term studies with people
      - And can we trust self reported calory intake?
    - Results in other organisms look impressive, e.g., mice (see chart)

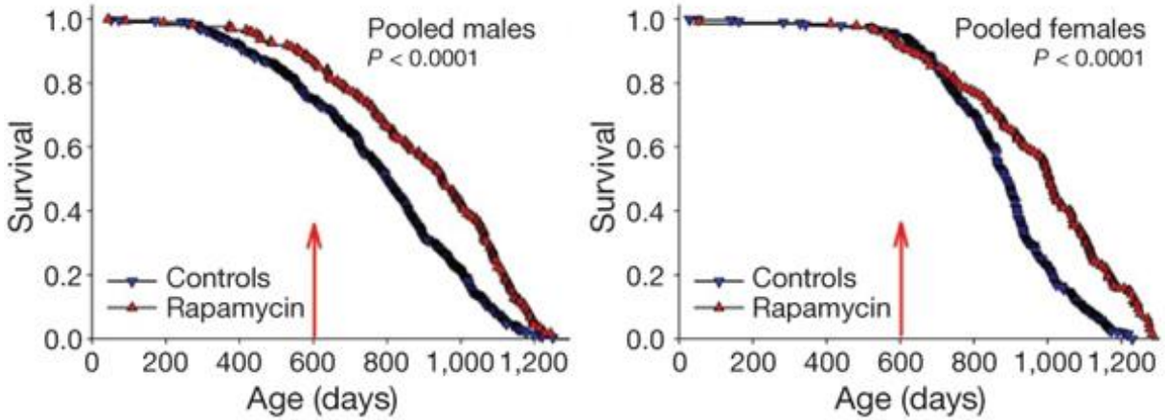


Source: Weindruch R, Sohal RS. Caloric intake and ageing. N Engl J Med. 1997 Oct 2;337(14):986-94. doi: 10.1056/NEJM199710023371407. PMID: 9309105; PMCID: PMC2851235.

Currently discussed approaches to influence the ageing process

Rapamycin:

- Inhibitor of the protein mTOR; numerous effects in the body, including improving autophagy ("cleaning up" damaged cells/cell components).
- Already approved for various purposes.
- Strongly (!) simplified: Rapamycin tricks the body into thinking it is under CR.
  - Hope: slowing ageing like CR but without its drawbacks.
  - Increase LE in mice (to lesser extent than actual CR but almost as high in some studies).



Source chart: Harrison, D., Strong, R., Sharp, Z. et al. Rapamycin fed late in life extends lifespan in genetically heterogeneous mice. Nature 460, 392-395 (2009). https://doi.org/10.1038/nature08221

Source of statement in conclusion box below: Juricic, P., Lu, YX., Leech, T. *et al.* Long-lasting geroprotection from brief rapamycin treatment in early adulthood by persistently increased intestinal autophagy. *Nat ageing* **2**, 824-836 (2022). https://doi.org/10.1038/s43587-022-00278-w

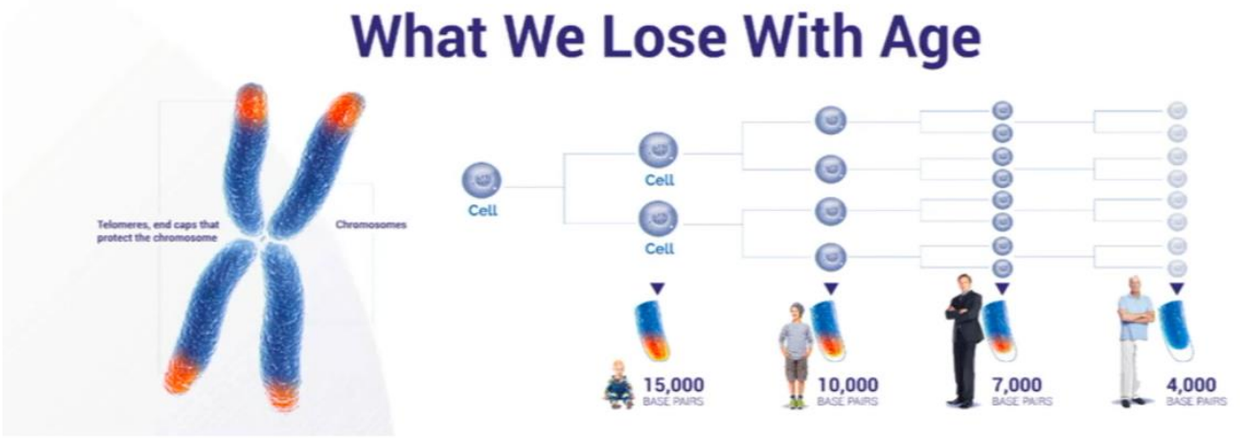
Effect on human ageing is currently being researched.

Already approved drug → "Safety" basically already tested. But: Permanent intake comes with side effects.
Current studies: Short-term use every now and then has a similar effect in mice as long-term use.

Currently discussed approaches to influence the ageing process

Telomerase (enzyme that lengthens telomeres):

- Telomeres shorten with each cell division
  - If they are too short, the cells can no longer divide (they die or become "senescent cells", see below).
- Telomerase counteracts this
  - significant successes in terms of lifespan in mice
  - in humans, it comes with an increased likelihood of cancer.



Source: Presentation: Novel Approach to Extending Human Healthspan - Insights from a Natural Experiment - Dr Douglas Vaughan at 2022 Fasano Longevity Conference; https://www.youtube.com/watch?v=ghOuNSHiOVU

My impression: After initial optimism, this topic became much quieter recently.

Currently discussed approaches to influence the ageing process

Backup

Senolytics (drugs for the elimination of senescent cells):

- In the course of life, senescent cells become more and more. The immune system becomes worse and worse and no longer manages to "clean them up".
  - Accumulation of these cells contributes substantially to ageing.
- Challenges:
  - How can drugs be made to attack only senescent cells (and not normal cells as well)?
  - Senescent cells can also have a benefit
- Currently great progress, just two examples
  - In senescent cells, "senescence-associated beta-galactosidase" is present. This "eats" a special type of sugar. If the senolytics are "packaged" in these sugars, they only reach the senescent cells.
  - Approaches to convert senescent cells back into "normal" cells (resveralogues).

Clearance of p16^{Ink4a}-positive senescent cells delays ageing-associated disorders

Darren J. Baker[1,2,3], Tobias Wijshake[1,4], Tamar Tchkonia[3], Nathan K. LeBrasseur[3,5], Bennett G. Childs[1], Bart van de Sluis[4], James L. Kirkland[3] & Jan M. van Deursen[1,2,3]

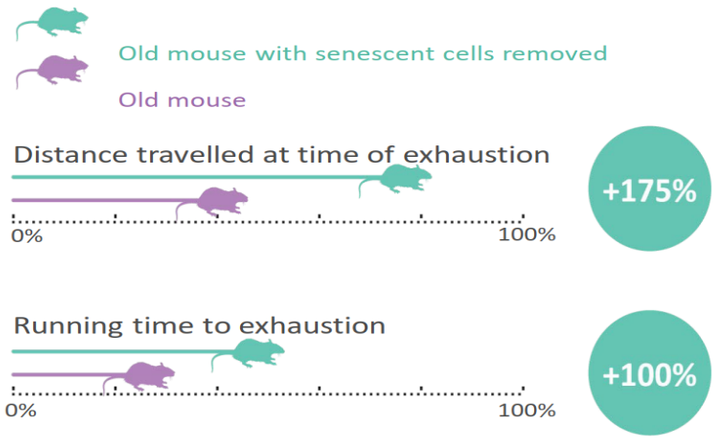
More than 60 ongoing human trials

ClinicalTrials.gov Search Results 07/07/2022

	Title	Status	Study Results	Conditions	Interventions
1	Senolytic Agents &Osteoarthritis	Not yet recruiting	No Results Available	•Osteoarthritis	•Drug: Quercetin Cap/Tab ,Fisetin Cap/Tab •Drug: Quercetin Cap/Tab,Fisetin Cap/ tab,Dioxynthizin capsules •Other: Placebo
2	Senolytic Agent Improve the Benefit of Platelet-Rich Plasma and Losartan	Recruiting	No Results Available	•Femoroacetabular Impingement	•Drug: Fisetin •Drug: Placebo
3	Use of Senolytic and Anti-Fibrotic Agents to Improve the Beneficial Effect of Bone Marrow Stem Cells for Osteoarthritis	Recruiting	No Results Available	•Osteoarthritis, Knee	•Drug: Fisetin •Drug: Losartan •Drug: Placebo - Losartan •Drug: Placebo Fisetin
4	Senolytic Therapy to Modulate Progression of Alzheimer's Disease	Active, not recruiting	No Results Available	•Alzheimer Disease	•Drug: Dasatinib + Quercetin
5	Senolytic Drugs Attenuate Osteoarthritis-Related Articular Cartilage Degeneration: A Clinical Trial	Active, not recruiting	No Results Available	•Osteoarthritis, Knee	•Dietary Supplement: Fisetin •Drug: Placebo oral capsule
6	Senolytic Therapy to Modulate the Progression of Alzheimer's Disease (STeMP-AD) Study	Recruiting	No Results Available	•Alzheimer Disease, Early Onset •Mild Cognitive Impairment	•Drug: Dasatinib + Quercetin •Other: Placebo Capsules
7	An Open-Label Intervention Trial to Reduce Senescence and Improve Frailty in Adult Survivors of Childhood Cancer	Recruiting	No Results Available	•Frailty •Childhood Cancer	•Drug: Dasatinib plus Quercetin •Drug: Fisetin
8	Senescence in Chronic Kidney Disease	Enrolling by invitation	No Results Available	•Chronic Kidney Disease	•Drug: Group 2: Dasatinib •Drug: Group 2: Quercetin
9	Cellular Senescence and COVID-19 Long-Hauler Syndrome	Recruiting	No Results Available	•SARS-CoV2 Infection	
10	Targeting Senescence to Reduce Osteoarthritis Pain and cartilaGE Breakdown (ROPE)	Not yet recruiting	No Results Available	•Osteoarthritis, Knee	•Drug: High-dose/short-duration Fisetin •Drug: Low-dose/sustained-duration Fisetin •Other: Oral placebo capsule
11	Targeting Cellular Senescence With Senolytics to Improve Skeletal Health in Older Humans	Recruiting	No Results Available	•Healthy	•Drug: Dasatinib •Drug: Quercetin •Drug: Fisetin
12	COVFIS-HOME: COVID-19 Pilot Study of Fisetin to Alleviate Dysfunction and Decrease Complications	Enrolling by invitation	No Results Available	•Covid19 •Coronavirus Infection	•Drug: Fisetin
13	COVID-FIS: Pilot in COVID-19 (SARS-CoV-2) of Fisetin in Older Adults in Nursing Homes	Enrolling by invitation	No Results Available	•Covid19 •SARS-CoV Infection	•Drug: Fisetin •Drug: Placebo
14	COVID-FISETIN: Pilot in SARS-CoV-2 of Fisetin to Alleviate Dysfunction and Inflammation	Enrolling by invitation	No Results Available	•Covid19	•Drug: Placebo •Drug: Fisetin

Rule of thumb: About 90% of trials fail. But this is now a numbers game...

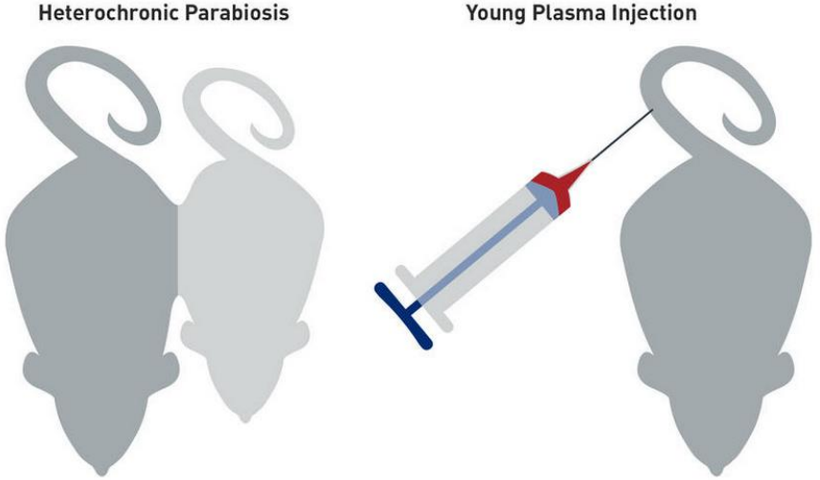
Source: Biology of Human Senescence, Talk of Richard Faragher, Professor of Biogerontology, at the SOA „Living to 100 conference", January 2023.



Currently discussed approaches to influence the ageing process

Heterochronic parabiosis and variations thereof (bloody science):

- The old mouse is rapidly getting better, the young one worse
- apparently both: “harmful” stuff in the old blood, and “useful” stuff in the young blood
  - not yet fully understood what exactly would need to be "filtered out" of the old blood
- It already seems to be beneficial to feed young blood plasma to old mice or to filter out old blood plasma and replace it with saline.
  - Very recent research: administration of bone marrow cells from young mice to old mice.



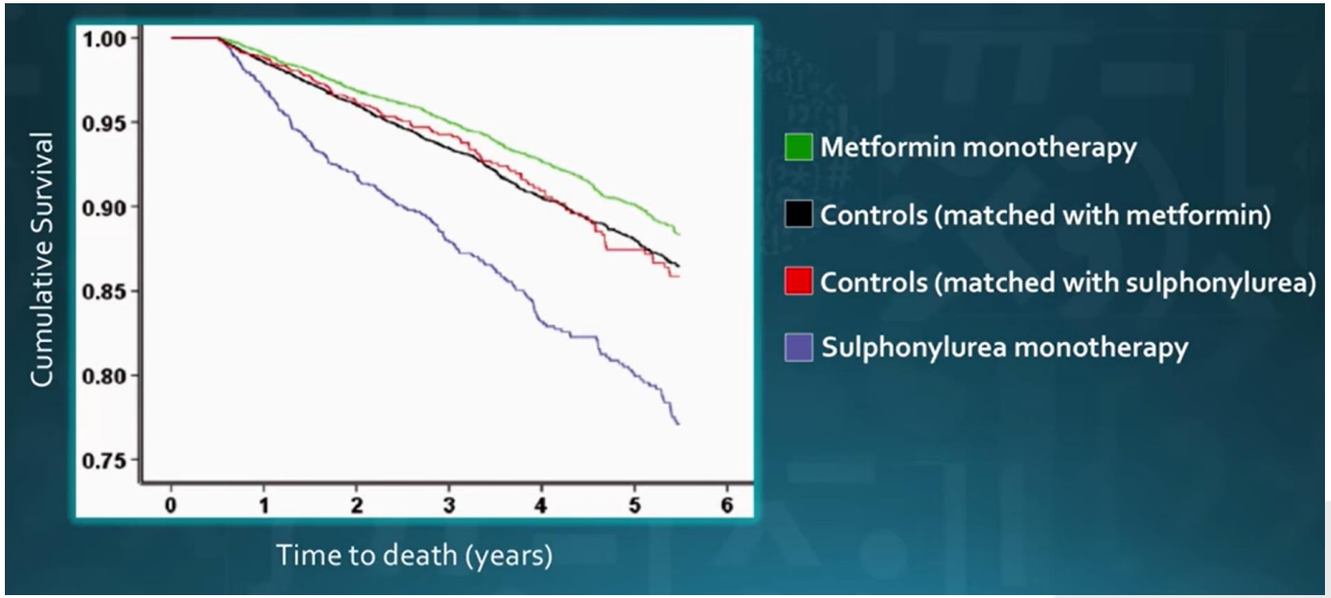
Source chart: https://www.jax.org/news-and-insights/2014/may/searching-for-the-secret-ingredients-of-the-fountain-of-youth

For more details, se, e.g., interview "Longevity update with Dr. Aubrey de Grey" https://www.youtube.com/watch?v=KEdRTzl2DgY starting at about minute 46 and publications of the researchers mentioned there.

Currently discussed approaches to influence the ageing process

Metformin:

- Standard diabetes drug. Diabetics who receive this, "age more slowly" (higher LE than non-diabetics!).



Ageing Research Reviews 40 (2017) 31–44

Contents lists available at ScienceDirect

Ageing Research Reviews

journal homepage: www.elsevier.com/locate/arr

Metformin reduces all-cause mortality and diseases of ageing independent of its effect on diabetes control: A systematic review and meta-analysis

Jared M. Campbell[a,b,*], Susan M. Bellman[a], Matthew D. Stephenson[a], Karolina Lisy.[c]

Metformin as a Tool to Target Aging

Nir Barzilai,[1,*] Jill P. Crandall,[1] Stephen B. Kritchevsky,[2] and Mark A. Espeland[2]
[1]Institute for Aging Research, Albert Einstein College of Medicine, Bronx, NY 10461, USA
[2]Wake Forest Older Americans Independence Center and the Sticht Center on Aging, Wake Forest School of Medicine, Winston-Salem, NC 27157, USA
*Correspondence: nir.barzilai@einstein.yu.edu
http://dx.doi.org/10.1016/j.cmet.2016.05.011

Aging has been targeted by genetic and dietary manipulation and by drugs in order to increase lifespan and health span in numerous models. Metformin, which has demonstrated protective effects against several age-related diseases in humans, will be tested in the TAME (Targeting Aging with Metformin) trial, as the initial step in the development of increasingly effective next-generation drugs.

Source: Can We Grow Older Without Getting Sicker? TEDMed Talk by Nir Barzilai, MD, AFAR Scientific Director and PI of the TAME Trial. https://youtu.be/MGKB9AdPmwc

Metformin currently probably most promising candidate for first medically based therapy against "ageing", cf. **TAME study**. Metformin was not chosen because it promised the greatest benefit, but because it has the lowest risk (safety already tested).

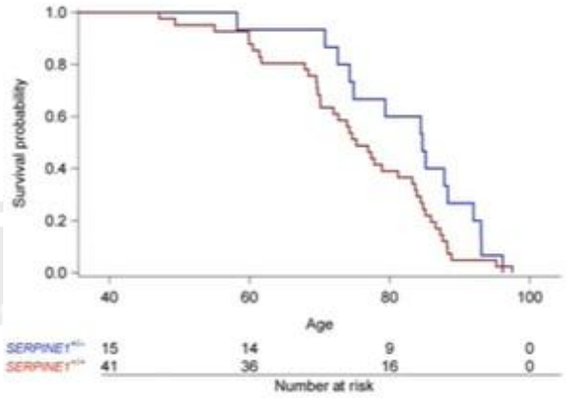
Currently discussed approaches to influence the ageing process

A null mutation in *SERPINE1* protects against biological aging in humans

Sadiya S. Khan,[1,2,3]* Sanjiv J. Shah,[1,2]* Ekaterina Klyachko,[1,2] Abigail S. Baldridge,[3] Mesut Eren,[2] Aaron T. Place,[2] Abraham Aviv,[4] Eli Puterman,[5] Donald M. Lloyd-Jones,[1,3] Meadow Heiman,[6] Toshio Miyata,[7] Sweta Gupta,[6] Amy D. Shapiro,[6] Douglas E. Vaughan[1,2†]

PAI-1 = protein that delays the dissolution of blood clots (plasminogen activator inhibitor-1; aka SERPINE1).

- Insights from an Indiana Amish community
  - Woman with strange disease: not a bleeder, but: After injury bleeding stops normally, after 1-2 days it starts again. Reason: anomaly in a gene that controls PAI-1 level - she has too little PAI-1.
- Douglas Vaughan became curious because he had done research on related proteins and knew that reduced PAI-1 levels in animals increased life expectancy.
  - because the Amish live relatively isolated: little genetic variation
  - A member who entered the community at the end of the 19th century brought this gene anomaly with him.
  - He had 17 children and 142 grandchildren. Today, about 15% of the 20,000 members have this gene anomaly and the reduced PAI-1 level (only very few in connection with the above-mentioned disease).
- Findings from comparison of community members with and without gene anomaly:
  - Those with gene anomaly (i.e., with the lower PAI-1 levels)
    - Have longer telomeres
    - Not a single person one of them has diabetes (in the comparison group, analogous to the total population)
    - Have higher lx; (but: small sample size!)



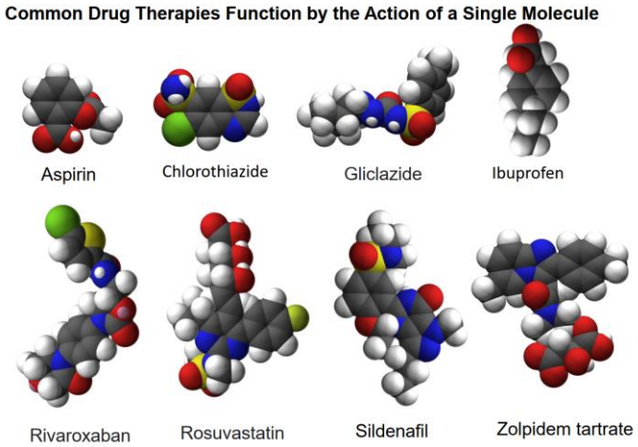
- There are already phase 3 trials on pills that lower PAI-1 level. (TM5614)

Source: Presentation: Novel Approach to Extending Human Healthspan - Insights from a Natural Experiment - Dr Douglas Vaughan at the 2022 Fasano Longevity Conference. https://www.youtube.com/watch?v=ghOuNSHiOVU

Currently discussed approaches to influence the ageing process

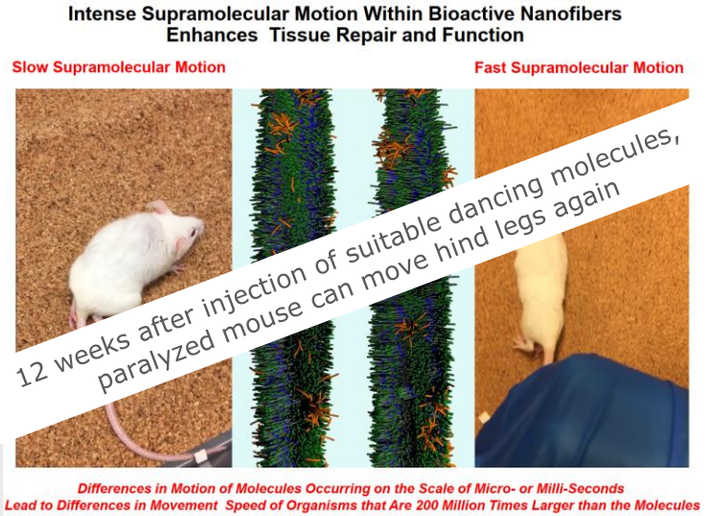
Backup

Dancing molecules (supramolecules activate processes in the body by sending a variety of signals → possible therapies in regenerative medicine).



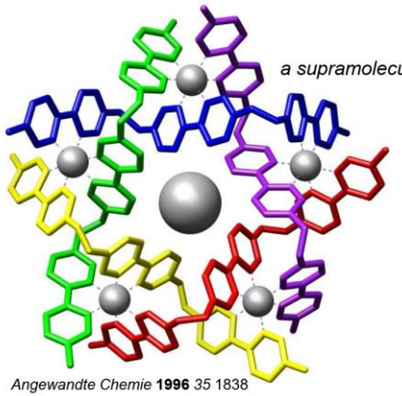
Application examples:

- BMP-2 ("bone morphogenetic protein") is used for regeneration of bones and cartilage; massive side effects possible in the necessary doses. In combination with signals from supramolecules, the dose can be greatly reduced.
- Special supramolecules (dancing molecules); "dancing movements" increase likelihood of signal reaching receptor. Can "repair" injured spinal cord.



Supramolecular Chemistry

chemistry "beyond" the molecule begins in the 1990s



a supramolecular structure

Jean-Marie Lehn
Nobel Prize in Chemistry 1987

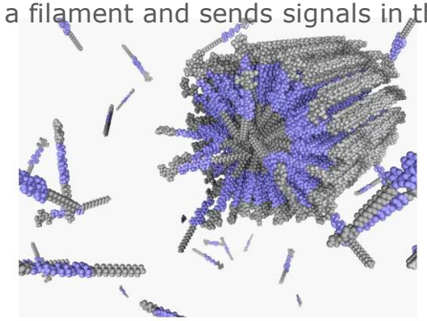
for the concept that molecules can engage in specific interactions as a result of their structure

Angewandte Chemie **1996** *35* 1838

could there be supramolecular therapies?

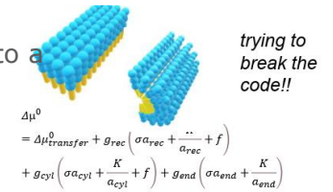
the therapy has many molecules, could be very potent and solve complex problems!!

molecule, which has been designed to self-assemble into a a filament and sends signals in the process.



200 nm

self-assembly into filaments driven by formation of Internal β-sheets or pseudo β-sheets



trying to break the code!!

$$\Delta\mu^0 = \Delta\mu^0_{transfer} + g_{rec}\left(\sigma a_{rec} + \frac{\ddot{}}{a_{rec}} + f\right) + g_{cyl}\left(\sigma a_{cyl} + \frac{K}{a_{cyl}} + f\right) + g_{end}\left(\sigma a_{end} + \frac{K}{a_{end}}\right)$$

Science **2001***294* 1684
Science **2004** *303* 1352
Science **2008** *319* 1812
Science **2010** *327* 555
Science **2012** *335* 813
Nature Materials **2016** *15* 469
JACS **2017** *139* 8915
Science **2018** *362* 808

Source: Presentation "Use of Dancing Molecules to Reverse Paralysis After Spinal Cord Injuries," Dr Samuel Stupp at 2022 Fasano Longevity Conference https://youtu.be/mG7M2VzZaX0

ifa

Part 3: Stuff that seems important to me but doesn't fit into any other chapter.

Future developments could happen much faster than before.

Epigenetic Clock

- DNA methylation measures the "true age of the body".
  - Epigenetic Clock
- Pioneer of this research: Steve Horvath
  - Requires DNA methylation at (only) about 1,000 genes
  - Calculates from this with a regression model the so-called Grim Age
    - "Works" to predict individual LE in humans and 120 other mammals.

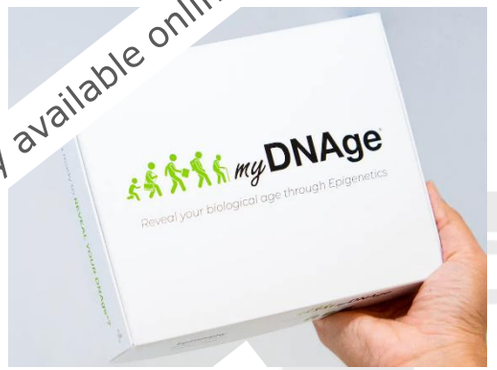
www.aging-us.com AGING 2019, Vol. 11, No. 2

Research Paper

DNA methylation GrimAge strongly predicts lifespan and healthspan

Ake T. Lu[1], Austin Quach[1], James G. Wilson[2], Alex P. Reiner[3], Abraham Aviv[4], Kenneth Raj[5], Lifang Hou[6], Andrea A. Baccarelli[7], Yun Li[8], James D. Stewart[9], Eric A. Whitsel[9,10], Themistocles L. Assimes[11,12], Luigi Ferrucci[13], Steve Horvath[1,14]

already available online



if you really, really love your dog ☺

Hope (greatly simplified): In the future, clinical trials will no longer have to wait for many years to see how a treatment changes mortality. Rather, we will be able to measure how the grim age (or another epigenetic clock) changes after a rather short period of time.

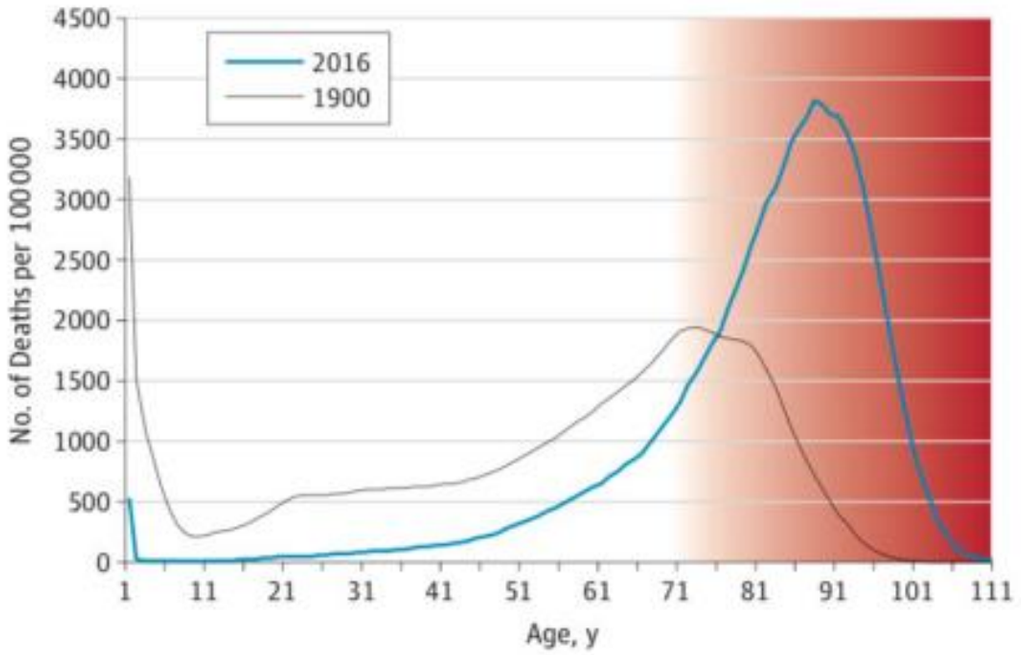
But also: Asymmetric information and resulting adverse selection in annuity markets?

The most optimistic player in this game

- He proposed the above-mentioned 7 „deadly things".
- probably overoptimistic in the presentation of his theses
  - e.g. Longevity escape velocity
  - e.g. LE 1000
  - e.g. "First person to live to 200 is already alive".
- But: serious research
  - SENSE
  - LEV Foundation
    - Currently investigating different combinations of rapamycin, senolytics, telomerase and variants of heterochronic parabiosis

Why everyone is talking about healthspan

Figure. Age Distribution of Life Table Deaths for Women in the United States, per 100 000 People, 1900 and 2016



The red zone represents a period in life when the risk of frailty and disability begins to increase rapidly. The goal of aging science is to delay and compress the red zone, which may extend healthy life. Sources: 1900 data from Bell and Miller[1]; 2016 data from Human Mortality Database.[2]

- Narrative:
  - Past: Survival pushed into the "red zone
  - Future: pushing the "red zone" out to higher ages
- Claim (not correct in my opinion):
  - This will result in an increase of (almost) only healthspan and hardly any lifespan.

Source: Keynote presentation by S. Jay Olshansky on the panel discussion "Futurism - Medical Knowledge of ageing" at the 2023 "living to 100" Symposium.

Part 4: Consequences of this uncertainty for measuring, modelling and managing (long term) longevity risk.

Preliminary note: MMM → UMMM

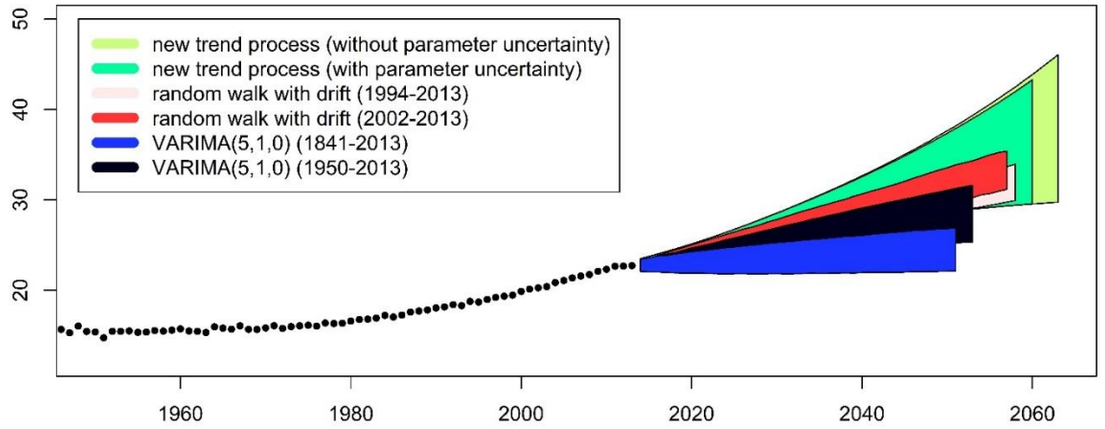
Natural cascade for dealing with risks

- Understand
- Model
- Measure
- Manage

It is often assumed, that the structure of a risk is understood. However, this is not so clear, with respect to longevity risk.

- If there are aspects that increase future uncertainty but are not observable from past data, calibrating the "dispersion parameters" of a model to historical data may not be appropriate, see below.
- If the facts clearly indicate that uncertainty is (much) higher over long periods than over short periods, one should (at least for modeling long-term risks) only use models that exhibit this property (see chart on the right).

Example: A mortality model calibrated to historical data. Then the parameters of the model are projected stochastically into the future with different processes. Graph: possible range for future "e60".



(Only) processes that allow for future trend changes meet the requirement that risk is higher for long periods of time.

- Estimate required for probability and intensity of trend changes.

Source: Matthias Börger, Johannes Schupp: Modeling trend processes in parametric mortality models. Insurance: Mathematics and Economics, Volume 78, 2018, Pages 369-380,

Possible approaches to dealing with these findings include

1) Observe and evaluate current developments

A suitable institution within the insurance and pension industry should

- ... systematically monitor developments
- ... assign a (qualitative) estimate to the developments (short-term vs. long-term; high vs. low proability; high vs. low impact if successful, etc.).
- ... present the results to market participants in a comprehensible form.

The question of whether these findings give rise to a need for action on the part of each company (pension fund / annuity provider) must of course be answered individually for each company.

2) Derive scenarios (understanding and modeling risks)

In my opinion, it would be desirable to derive scenarios that could be used for scenario analyses and also as "calibration supports" for stochastic models.

- E.g., optimistic scenario: drug A comes to market in 10 years, drug B in 15 years
  - What would the life expectancy be in this path?
- Analogously: pessimistic scenario: no breakthrough in slowing ageing, but climate change and microplastic
  - What would the life expectancy be in this path?
- Possible benefit
  - Use of these scenarios in scenario analysis
  - If statements of the following type can be made:
    - "The probability that things will be even better (worse) than in the optimistic (pessimistic) scenario is x% (y%)"
  - Then stochastic models can be calibrated accordingly.

Interdisciplinary cooperation required!

Possible approaches to dealing with these findings include

3) Risk analyses (measure risks)

With the scenarios and models derived in 2), the risks of a company (e.g., pension fund) can be analyzed.

In particular, this allows an assessment whether previous risk management measures are sufficient.

4) Derive options for action (manage risks)

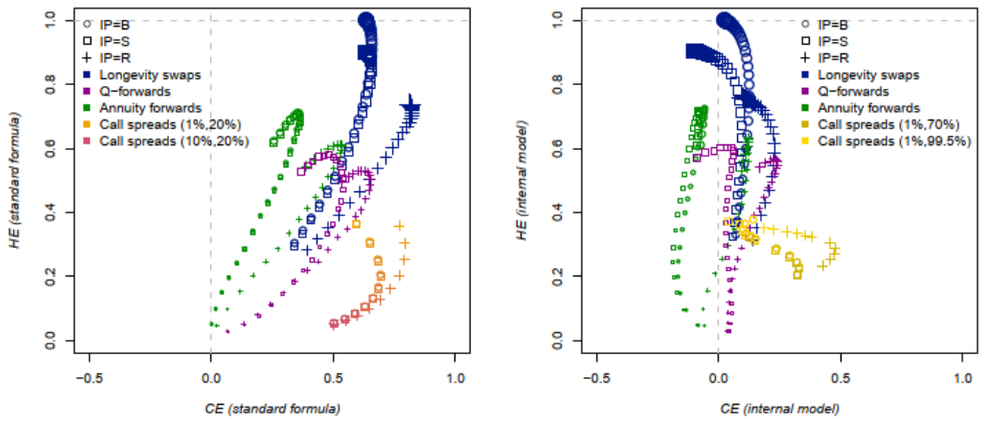
Options for, e.g., an annuity provider could be:

- No change required.
- No longer offer lifelong annuities
- Product design with lower “systematic longevity risk” for the insurer
  - weaker guarantees
  - tontines
  - etc.
- Hedging of longevity risks (see chart on the right)

There are numerous different hedging instruments (mortality derivatives), which differ greatly in their effect.

When dealing with this issue, it is essential to analyze the following questions simultaneously:

- What's the impact? (Risk reduction)
- What does it cost (price of the hedging instrument, for insurers also savings on the cost of capital)
  - If you believe that "the market" also underestimates uncertainty: "Price<Value".



(a) Standard formula (b) Internal model

Source: Börger, M., Freimann, A., and Ruß, J. (2021). A combined analysis of hedge effectiveness and capital efficiency in longevity hedging. Insurance: Mathematics and Economics, 99:309–326.

A brief conclusion and a thesis

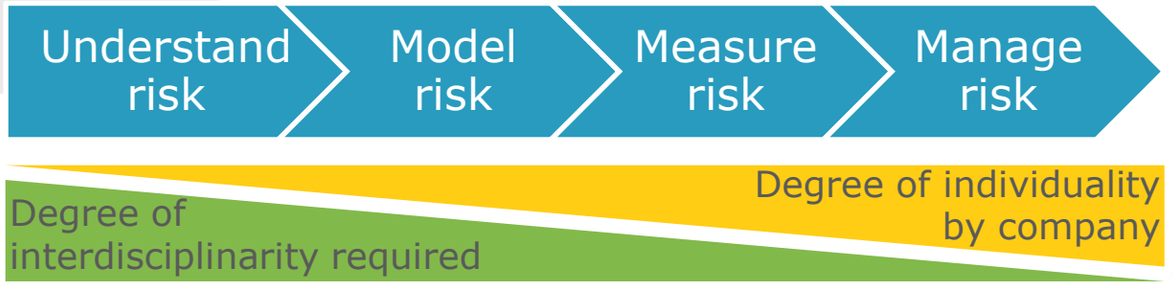
A brief conclusion and a thesis

Conclusion: With regard to the future development of life expectancy, changes are possible in both directions.

- The aspects that argue for a slowdown in the increase in life expectancy are already present. A dampening seems very likely in the near future (and can already be observed in some countries).
- The slowing of the human ageing process could (in a more distant future) lead to a rather large increase in human life expectancy.
  - No one can seriously predict today if and when "something will happen" and how big the impact will be.
  - However, the sheer number of possible therapies, some of which are already very advanced, suggests that something will happen in the next few decades with a probability that is clearly > 0 (but not 100%!!!).
  - That's uncertainty!
- In particular: This can become relevant before a typical insured person buying a deferred annuity today will have died.

Thesis: The insurance industry must pay more attention to this topic in the future than in the past.

- **Individual view:** Uncertainty of individual life span (which is usually even more uncertain than life expectancy) is much larger than most people think.
  - Managing this uncertainty through lifelong income (buying an annuity) is becoming increasingly important.
- **Collective view:** The so-called systematic longevity risk on insurers' books may be greater than assumed. Typical stochastic models might underestimate this risk.
  - More innovative risk management measures could gain importance (reinsurance, "longevity derivatives", alternative product design (e.g. tontines),...).



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