

AI in Longevity: How Artificial Intelligence Is Discovering Age-Delaying Medicines

Introduction

Ageing is considered to be the most significant risk factor for the most prevalent diseases of developed countries: cancer, cardiovascular diseases and neurodegeneration (Niccoli and Partridge, 2012). Yet, many in the general public and the scientific community accept it as an inevitable, natural process that we should not interfere with. But if it is the underlying cause of so many pathologies, why not treat it as a disease? And if we do consider it to be one, can we one day cure it?

These are the questions that have preoccupied the brightest minds in longevity and geroscience for decades. In recent years, studies have uncovered many causal mechanisms of ageing and discovered possible interventions to slow down, or even reverse, its progression. Among these, some pharmaceutical interventions, such as caloric restriction mimetics and senolytics, are showing some encouraging results (Hassani *et al.*, 2022)

In the era of technological advancement, artificial intelligence (AI) is transforming the field of biomedical research. Many AI tools are utilised to find, predict, analyse and evaluate new medical interventions with unmatched speed and efficiency. Longevity scientists are embracing innovations to identify longevity- and healthspan-promoting molecules. This essay explores the past, present, and future uses of AI in the discovery of such compounds.

The Science of Ageing

Before jumping into the discussion, it is essential to establish what is meant by 'ageing'. A person's chronological age is simply the time passed since their birth, whereas the term 'biological ageing' refers to the more complex concept of accumulation of cellular damage over time that leads to the deterioration of the individual's physiological state, which increases vulnerability to disease and death. The two types of ageing may occur at different

paces for the same individual, as unlike chronological age, biological ageing is impacted by factors such as lifestyle, diet, genetics and environment (Morales-Brown, 2025).

Scientists have discovered nine of the most prominent and reliable hallmarks of biological ageing, and they are theorised to not only indicate but also cause ageing. Those are epigenetic alterations, telomere attrition, genomic instability, cellular senescence and others presented in Figure 1. The hallmarks are all interconnected and occur during both ‘normal-paced’ and accelerated ageing (López-Otín *et al.*, 2013). Biological ageing is often quantified by ‘epigenetic clocks’ that estimate biological age by measuring the person’s DNA methylation levels. DNA methylation is the addition of methyl tags (-CH₃) to DNA sequences, which regulates gene expression, and the patterns of these tags change in a predictable manner with age (Moore, Le and Fan, 2012). By analysing methylation at specific, ageing-related genomic locations, an individual’s biological age, which can also be referred to as ‘epigenetic age’, can be estimated with great accuracy (Horvath, 2013).

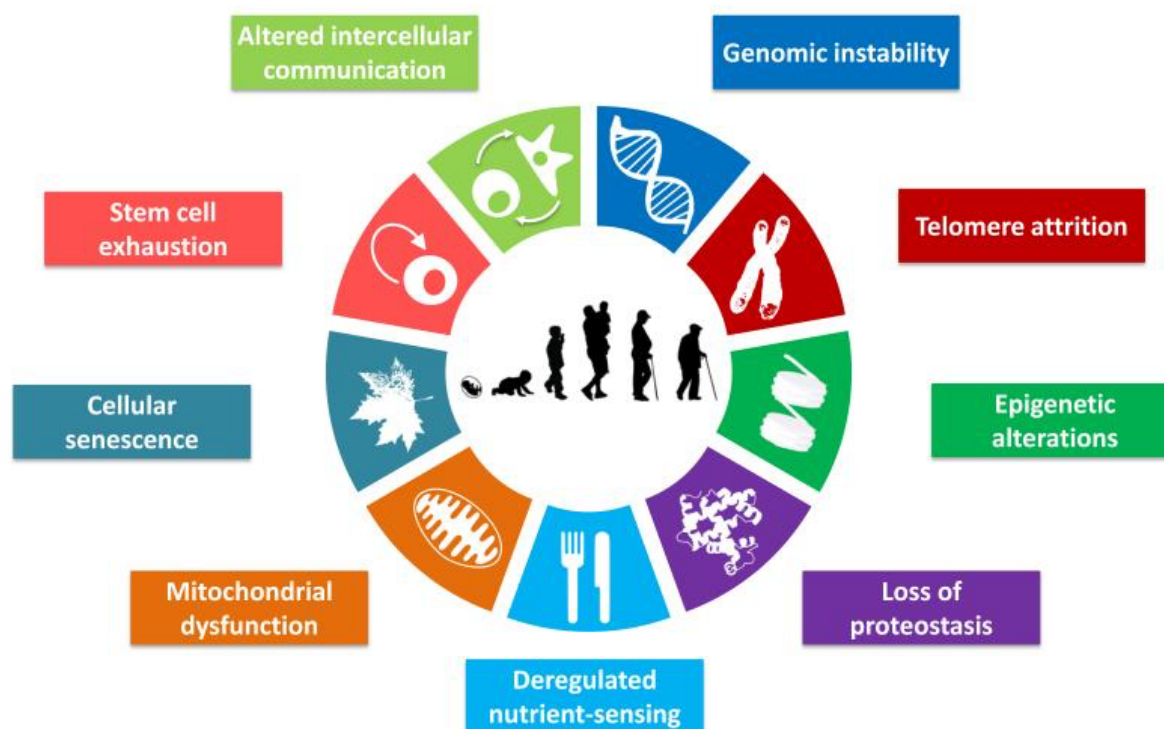


Figure 1. Diagram displaying the nine hallmarks of ageing (López-Otín *et al.*, 2013).

Existing Anti-Ageing Compounds

There are many molecular pathways underlying ageing, and scientists are developing therapies, such as pharmaceuticals or lifestyle interventions, that target those pathways to slow down or reverse ageing. One of the more famous types of medicines used to promote longevity is caloric restriction mimetics. Caloric restriction (CR) is a reduction in caloric intake without malnutrition, and it has been shown to prolong the lifespans of many common model species used in longevity research, such as mice, yeast and roundworms (Madeo *et al.*, 2019; Andrea Di Francesco *et al.*, 2024; Leonov *et al.*, 2017; Smith *et al.*, 2008). The first controlled study of the effects of long-term CR on longevity of humans ‘CALERIE’ showed a decrease in the pace of ageing, as measured by Dunedin PACE epigenetic clock (Ryan *et al.*, 2024).

Geroprotectors are a class of molecules that are capable of slowing the processes of ageing in model organisms (Moskalev *et al.*, 2017). Many compounds have been found to target molecular pathways inhibited or activated during CR. One of the more studied longevity medicines is rapamycin, an antifungal and immunosuppressive compound discovered in the 1970s from *Streptomyces hygroscopicus* on Easter Island (Roark and Iffland, 2025). It suppresses mTOR (mechanistic Target of Rapamycin), a nutrient and energy-sensing pathway that regulates growth and metabolism, which affects many hallmarks of ageing (Stallone *et al.*, 2019). Rapamycin has displayed an extension of lifespans of mice, fruit flies, nematodes and yeasts (Harrison *et al.*, 2009; Bjedov *et al.*, 2010; Zhang *et al.*, 2024; Powers, 2006). However, it has been noted that prolonged use of rapamycin could lead to liver damage, inflammation and tumorigenesis, which highlights the need for the discovery of safer compounds that target mTOR (Umemura *et al.*, 2014).

Metformin, a common and safe type II diabetes medicine, has shown to activate AMPK, which reduces inflammation, inhibits mTOR and enhances autophagy and DNA-repair, which in turn prolong longevity. The drug was discovered way back in the 17th century from a French lilac *Galega officinalis*, which contains metformin-like guanidine compounds that regulate blood glucose levels (Soukas, Hao and Wu, 2019). The geroprotective effects of

metformin mimic those of dietary restriction, and diabetic patients taking the drug have observed a reduced incidence of cancer, cardiovascular disease, and overall mortality (Barzilai *et al.*, 2016).

An important therapeutic longevity target is cellular senescence. Senescent cells, sometimes called zombie cells, are cells that exited the cell cycle without undergoing apoptosis (cell death), and continue to secrete inflammatory compounds (named SASP (senescence-associated secretory phenotype)) that can damage neighbouring cells (as shown by Figure 2). They have an important role in embryonic development, childbirth, and wound healing; however, their accumulation contributes to many age-related diseases and, as a result, is considered a hallmark of ageing (National Institute on Aging, 2021).

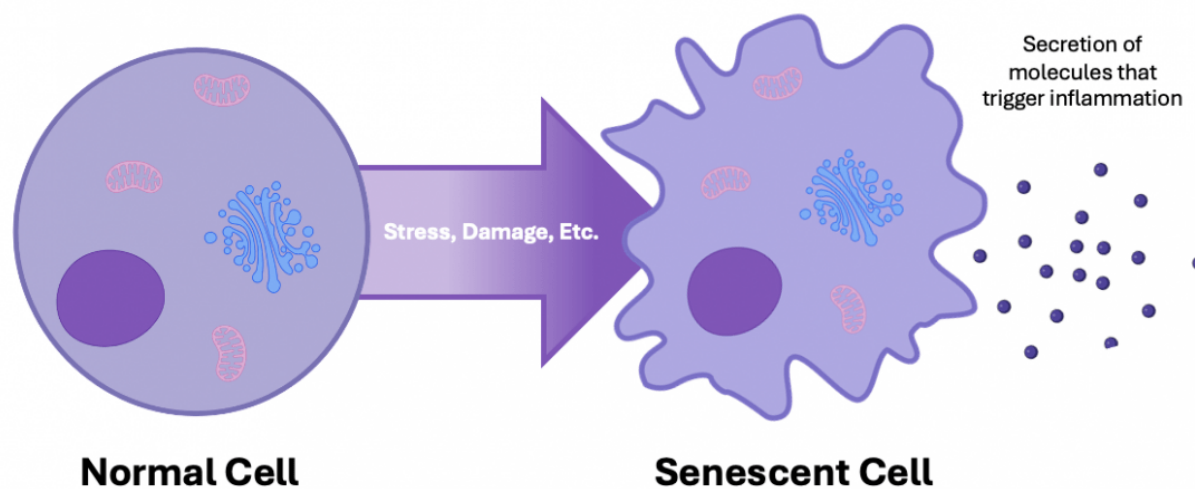


Figure 2. Diagram illustrating cellular senescence (Kalser, 2024).

Senolytics are compounds that selectively kill senescent cells by disabling their survival networks called SCAPs. Senolytics can temporarily disable SCAP networks to allow apoptosis. (Hickson *et al.*, 2019). These compounds were discovered by working backwards from the identification of these networks: the researchers proved the vitalness of SCAP for

senescence and repurposed existing pharmacology to target it. A study testing a combination of senolytics – Dasatinib and Quercetin – showed a prolonged healthspan and delayed onset of age-related diseases in mice (Zhu *et al.*, 2015).

All of the previously mentioned medicines demonstrate the possibility of treating ageing pharmacologically. However, their discovery relied on observations and classical biochemical approaches. These are costly, slow, and based on trial-and-error in wet laboratories. Because there is a clear need for designing and testing new, safer and more effective compounds to extend lifespan, AI has been employed to accelerate this process.

AI and Biomedical Research

Artificial Intelligence (AI) is a broad term for novel technologies that simulate human intelligence to perform tasks like problem-solving and decision-making. This field of science was formally established in 1956 at the Dartmouth Workshop, organised by John McCarthy (Tableau, 2025). There are various subsets of AI, such as machine learning and deep learning, that are frequently utilised for medical research. Machine learning (ML) is a computer system that can “learn and improve from experience without being explicitly programmed”, whereas deep learning (DL) is a subfield of ML that uses artificial neural networks to learn from large datasets (University of North Florida, Office of Faculty Excellence, 2025).

AI is used by researchers to identify patterns and correlations within enormous, complex datasets, such as genomic and proteomic data. These datasets are very difficult to analyse, and the specific relationships can often be missed by the human eye due to the large number of variables. Employing AI for these tasks not only accelerates the process but also leads to new therapeutic targets that would not have existed without it. (Cook, 2024).

Moreover, deep learning was used by Google DeepMind to develop a system to predict the three-dimensional structure of a protein from its amino acid sequence, called AlphaFold (Jumper *et al.*, 2021). This technology has many implications in biomedical research, such as

studying pathologies associated with protein misfolding and dysfunction (including cancer and rheumatoid arthritis), synthesising novel proteins with specific functions to treat diseases, and examining the impact of DNA and mRNA mutations on proteins' structure and stability (Geek Nomad, 2024; Pak *et al.*, 2023).

Many other implications of AI include diagnostic imaging (for diseases such as skin cancer) (Frasier *et al.*, 2025), helping healthcare professionals better diagnose patients by providing a second opinion, predicting pathologies like sepsis (Haas and McGill, 2022), and countless others. AI, with its ability to analyse giant datasets in seconds, is transforming the efficiency and accuracy of research in all aspects of science. It has been especially useful in identifying, predicting and analysing novel medicines against countless illnesses. Naturally, the field of longevity is not behind in the use of this powerful tool.

AI in Longevity Research

This section will explore the implications of AI in identifying biological pathways to target with medicines, generating new or repurposing existing compounds for pro-longevity purposes, as well as validating and testing them for clinical use.

a. AI and Target Identification

The complicated nature of ageing biology (involving interconnected pathways such as previously mentioned mTOR, AMPK, senescence and many others) traditionally required years of laboratory research to find specific druggable targets. This heterogeneous cause of age-related diseases often leads to poor efficacy of existing interventions, in part because of inadequate target choice or the inability to find the group of patients who respond best to the treatment (Zavoronkov, 2022). This is where AI can step in.

AI is increasingly being applied to multi-omics data to distinguish new longevity targets. Multi-omics data, consisting of genomic (whole genome sequencing), transcriptomic (RNA sequencing), proteomic (proteins and their structure) and metabolomic (end products of

cellular metabolism) data, change with age (Baião *et al.*, 2025). Software PandaOmics, developed by Insilico Medicine, identifies novel targets by applying deep learning models to this multimodal omics data, as well as evaluating background evidence to short-list the most promising targets (Kamya *et al.*, 2024).

b. Repurposing and Generating Compounds with AI

The approach of applying machine learning to complex data is also used to identify compounds that already exist to be repurposed to target ageing-associated pathways. In Ribeiro *et al.*'s (2023) study, the team trained a machine learning classifier on DrugAge data (a database of longevity compounds (Barardo *et al.*, 2017)) to predict not only the chemical structure of compounds but also the biological pathways and proteins targeted by the molecules. This model can tell whether a certain molecule has lifespan-extending properties, and demonstrates how AI can simultaneously identify geroprotective medicines and generate their mechanistic hypotheses, highlighting the great potential for repurposing molecules for longevity-related uses (Ribeiro *et al.*, 2023).

AI is also being used to generate new chemical compounds from scratch that exhibit pro-longevity properties. An example of this can be seen in Insilico Medicine, which employs deep learning at multiple stages of dual-purpose (ageing and age-related diseases) drug discovery. After identifying the biological target with PandaOmics, they employ the Chemistry42 platform, which uses a novel AI technique called generative adversarial networks (GANs) to create molecules that specifically work on the targets (Insilico Medicine, 2024; Diamandis, 2022). So far, they have successfully developed a TNIK inhibitor (TNIK is an enzyme involved in signal transduction, gene transcription, and cytoskeletal organisation (National Centre for Biotechnology Information, 2025)) that has been effective at targeting idiopathic pulmonary fibrosis in both pre-clinical and clinical models.

c. Testing and Validation with AI

At the very early stages of drug development, many *in silico* (on a computer or a virtual simulation) tests are conducted to try to predict the success of a certain molecule, prior to *in vivo* testing. A good example of this approach would be Insilico's InClinico AI platform, which can predict the probability of success of the individual trials of their newly generated compounds to assess whether they should be tested further (Insilico Medicine, 2020).

AI can also be used to develop new biomarkers to measure the rate of ageing, which is especially useful in trials of geroprotective molecules. The previously mentioned epigenetic clocks are statistical models that quantify the biological age of an organism; this is achieved by analysing DNA methylation levels at specific CpG sites (locations within the genome). In Dr. Steve Horvath's first-ever-created epigenetic clock (2013), he employed an elastic net regression ML algorithm to assign how much individual CpG sites correlate with ageing. In my own research project, I used a publicly available AI tool 'ChatGPT' to apply the Meir *et al.* (2018) epigenetic clock formula to a DNA methylation dataset of mice that underwent an endurance training program (as well as a control group) to investigate exercise's effect on ageing. Despite the investigation not focusing on pharmacological intervention, this exemplifies how AI improves the efficiency of research by conducting mathematical tasks to quantify the impacts of the intervention on ageing, as if I chose to perform the calculation by hand, it would have taken me significantly more time.

Lastly, AI platforms are embedded within feedback loops. Both positive and negative results from *in vitro* and *in vivo* trials are analysed by the AI algorithms that repurpose and generate new compounds to improve their future predictions through active learning.

d. Case-study: SYDRA Biotech

The co-founder of SYDRA, Dr. Alexander Dakhovnik, has kindly agreed to give me an interview about the company's work on developing geroprotectors using artificial intelligence. At SYDRA, they approach the process of drug discovery from a different angle –

instead of identifying a biological target, they begin with a molecule. They first use an artificial neural network (ANN) to identify the promising compound, then test it *in silico* and in *C. elegans* (roundworms), and only then start the process of pinpointing the biological target of the drug. As Dr. Dakhovnik stated in the interview, 'we decided to reverse engineer drug discovery – from phenotype to target, not from target to phenotype'.

The company has developed a novel AI molecule discriminator that predicts whether a compound has anti-ageing properties. Their ANN, essentially being an 'AI chemist', was first trained on a large database of small molecules and then fine-tuned on a dataset of geroprotectors. Using this tool, 11 molecules were identified as safe and likely to prolong longevity, 5 of which have shown lifespan prolongation of 6-14.5% in *C. elegans*. Some of them were then tested in mice, with 2 being particularly successful at lifespan extension, achieving an impressive median lifespan elongation of 23% and 29% (Dr. Alexander Dakhovnik, personal communication, August 2025).

The process of *in vivo* testing of the identified molecules at SYDRA happens in *C. elegans* model organisms and is optimised using the automated NemaBot roundworm lifespan assays. Instead of relying on manual monitoring, SYDRA employs AI to scan and analyse the worms' activity to detect their ageing progression with and without the pharmacological intervention (Dr. Alexander Dakhovnik, personal communication, August 2025).

A big challenge faced by SYDRA was the fact that ageing itself is not classified as a disease (yet), and therefore cannot be treated directly. As their main goal was to show the extension of lifespan, which Dr Dakhovnik believes to be the only valid indicator of true age-targeting, geroprotective interventions, to commercialise the discovered molecule they have to prove that the compound treats an FDA-approved age-related disease. To do this, they plan to employ whole-genome CRISPR-Cas screenings to identify which proteins were targeted, and therefore which age-related disease the compound might affect the most. This approach is called a target deconvolution.

They have also started to develop another AI model that generates completely novel molecules from noise to act as geroprotectors. They have partnered with Enamine to

synthesise those generated molecules to test them *in vivo*, which might produce promising results.

The Future of AI in Ageing

There is no doubt that artificial intelligence will play a larger and larger role in the years to come. Its use in longevity will decrease the drug discovery timeline from prediction to clinical testing, by both employing the previously mentioned approaches and discovering new ways to streamline the process, compressing the pathway to just a few years or even months.

One key future implication of AI is personalised medicine. By integrating deep learning models that work on even larger multimodal datasets (that might include not only omics data but also imaging, clinical history and other variables), the algorithms might be able to predict exactly how and which geroprotectors will most likely extend the patient's lifespan and help cure their age-related diseases.

However, there are some challenges to the AI-optimised drug discovery. A vital limitation is data bias, since most training data for AI may come from small and genetically similar cohorts, which may not represent the global diversity and better suit certain groups of people. A big hurdle is the regulatory institutions' (such as the FDA and EMA) slow adaptation to quick progress in AI-found molecules and AI-reliant trial protocols, as well as their reluctance to classify ageing as a disease. Due to this, it is difficult for researchers to trial their geroprotective molecules in *Homo sapiens*, which is vital for knowing whether they truly prolong human lives. Another problem that may arise is the accessibility of those novel medicines, as there is a risk that they will be expensive and not equally distributed, contributing to disparity within society. Nonetheless, the potential of AI scaling and shortening the molecule discovery process might reduce the costs, contributing to better equality.

Conclusion

As seen by examples provided in this essay, such as enhanced molecule generation and repurposing, advanced biological target identification, and validation process optimisation, AI is already accelerating drug discovery at unmatched speeds. The future of the industry seems incredibly promising, with countless possible new uses of AI in the field and many yet-to-come medicines potentially extending the lifespans of millions of people.

The biggest takeaway from this is that AI is not here to replace hardworking scientists – it is a powerful tool that only works alongside qualified specialists to supercharge and, in the case of SYDRA, completely change the traditional drug discovery pipeline to open doors to many novel pharmacological interventions that can prolong healthy human lives.

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