Longevity Research Tsunami

BY WILLIAM FALOON

For nearly 50 years, I've professionally advocated that substantial resources be allocated to support **longevity research**.

My early talks/articles were met with skepticism by those who could not conceive of older people growing biologically *younger*.

In recent decades, scientific advances have enabled me to pursue and directly fund a concept that:

Age reversal may become medically achievable within our lifetime.

My recent lectures at biomedical conferences emphasize a convergence of technologies that are changing **aging research** into an exciting **bioengineering** endeavor.

Promising solutions include partial **cellular reprogramming**, senescent cell clearance, AI-driven drug discovery, organ regeneration, and therapies aimed at restoring ***youthful*** functionality.

While scientists remain cautious about timelines, I view laboratory developments occurring today as beyond *anything* previously seen in medicine.

This editorial describes a few topics from my recent live presentations on the science behind **biological age** reversal.

Cellular Reprogramming

My research team, along with others, is funding research that adds copies of **genes** that have been shown to improve health and/or longevity in **old cells** and **old mice**.

In recent years, my presentations describe the discovery that **aged cells** may be pushed back toward a *younger* **biological** state[1] through the use of **reprogramming** factors first identified by Dr. Shinya Yamanaka.[2,3]

These "**Yamanaka factors**" — often abbreviated as **OSKM** or **OSK** — demonstrate that mature (old) cells can be reverted to a more *youthful* state.[4]

Note that **OSK** is the abbreviation for three of the four Yamanaka transcription factors.

Yamanaka factors are one of the most important discoveries in modern biology.

Research around these factors suggests **aging** may not simply be passive wear and tear, but a **reversible** process via **genetic optimization**.

In this editorial, I highlight findings from experiments in which **old mice** experienced restoration of tissue function, improved regeneration, and/or extension of remaining lifespan after **partial cell reprogramming**.[1,5,6]

If restoring the *youthful* identity of your **old cells** by resetting your **epigenome** seems speculative, understand that the **vitamin D** you take is known to directly affect at least **hundreds** of genes, and indirectly affects thousands more.

It both upregulates and downregulates target genes involved in diverse physiological processes.[7-9]

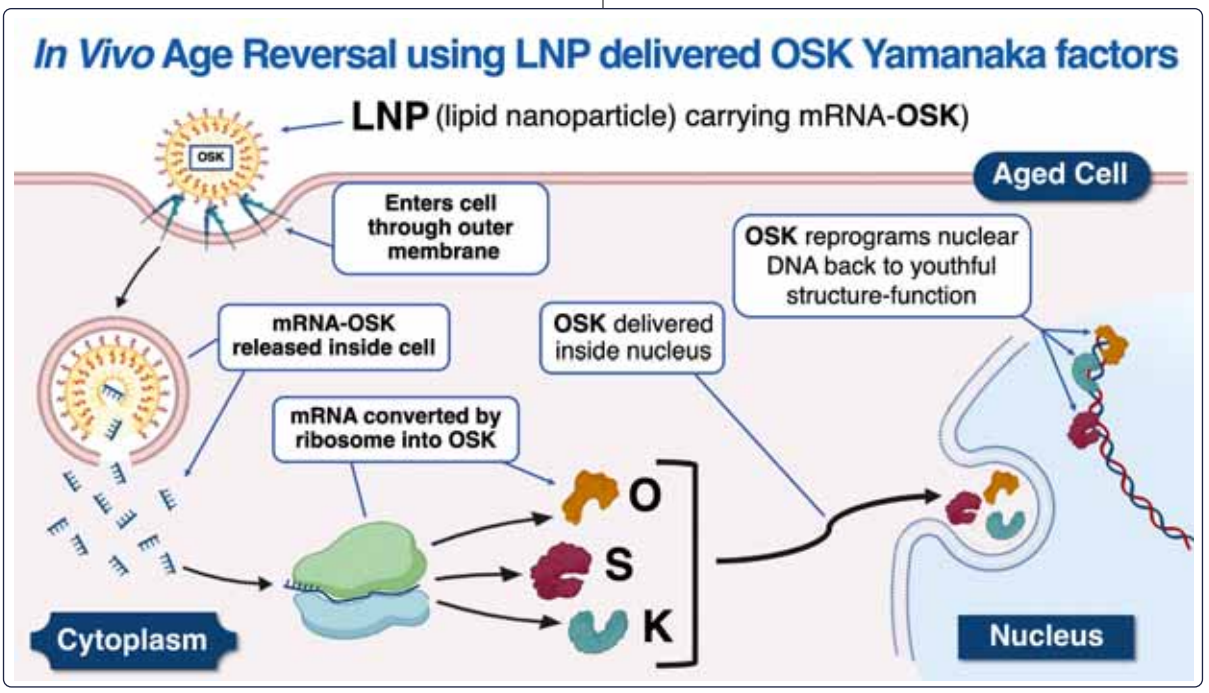
For those who are not familiar with the term **epigenome**, this is a set of mechanisms inside cells that regulate which **genes** are turned "on" and "off".[10]

(Note: While **vitamin D** beneficially impacts gene expression, it does NOT perform **cellular reprogramming** in the same sense as epigenetic reprogramming observed with Yamanaka factors.)[9]

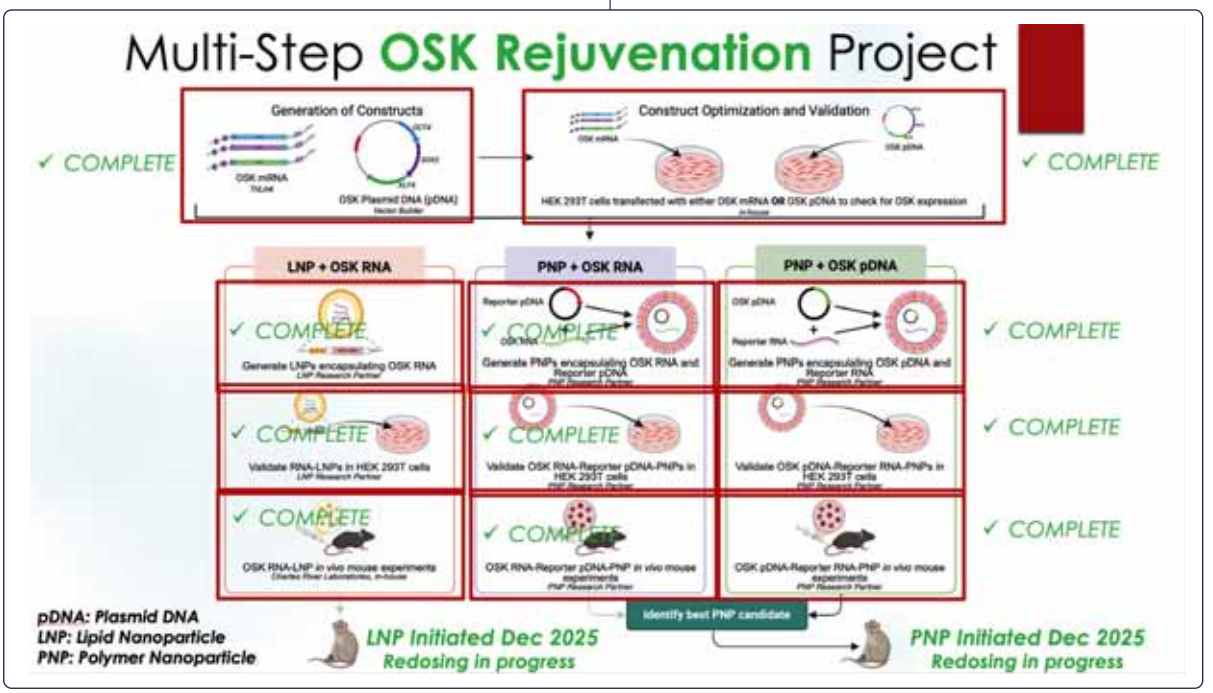
Rather than viewing **aging** as an irreversible accumulation of damage, I present evidence showing our body may be capable of restoring *younger* functions if properly activated via **cellular reprogramming** and other interventions.

The graphic on this page is what I present at scientific conferences to depict delivery of **OSK** into nuclear DNA, where it can **reprogram** old cells back to a more youthful state, as shown in preclinical studies.

Our research team is funding a study using OSK *in vivo* in an attempt to **reverse** aging in old primates.



See how we encase the **OSK** RNA blueprint into a lipid nanoparticle to enable it to easily cross the cell membrane. Once inside, the lipid nanoparticle releases the OSK blueprint that is converted to OSK proteins by ribosomes. These OSK proteins then pass through pores in the nucleus where they bind to nuclear DNA and induce a "**reprogramming**" effect that enables old cells to regain *youthful* structure-function.[11]



Cellular Reprogramming: From Petri Dish to Preclinical Validation

Preclinical evaluation pipeline for OSK-based reprogramming constructs, including in vitro validation, in vivo testing in mouse models, and subsequent evaluation in non-human primates.

Measuring Biological Age

Starting in the mid-**1990**s, our **Life Extension**® group attempted to develop tests to assess **biological age**.

We knew that, to rapidly determine whether a therapy might extend a healthy lifespan, we required a way to determine if experimental **interventions** reduce **biological age** markers.

The state of biomedical technology in that era prevented us from achieving consistent results.

But fortunately, starting in 2011, other research groups succeeded in developing **DNA methylation**-based age measurement tools using saliva[12] and blood samples, as well as a composite **phenotypic age** score derived from existing blood tests such as **C-reactive protein** (CRP), **glucose**, **immune cells**, etc.[13]

These transformative tools (and other tests) allow scientists to quantify aging in ways that were impossible decades ago.

Instead of waiting years to determine whether a therapy extends lifespan, researchers can now examine whether interventions appear to reduce biological **age** markers in months or even weeks.

Counteracting Other Aging Mechanisms

We are funding an expensive primate project using nanoparticle-delivered **OSK gene** addition therapy.

We are simultaneously studying other interventions with significant regenerative potential.

I've enlightened audiences about **senescent cells** that secrete destructive compounds into surrounding tissue. Senescent cells contribute to frailty, fibrosis, vascular disease, osteoarthritis, and systemic **inflammation**.[14]

Even if **OSK** genes succeed in **reprogramming** old cells back to youth, we still may need to eliminate **senescent cells** using **senolytic** compounds such as fisetin, quercetin, and senolytic drugs that are being developed.[15]

Animal studies that use **senolytics** to clear senescent cells have shown improvements in physical function and an extended lifespan.[16] Senolytics may be analogous to periodically removing malfunctioning components from an aging machine. While human evidence remains limited, **senolytic** interventions could eventually become part of routine **preventive** medical practice.

Artificial intelligence-driven drug discovery offers tremendous potential.[17] I often discuss companies such as **Insilico Medicine**, which we helped **fund** in its early years.

Insilico has pioneered machine learning systems capable of identifying therapeutic compounds at unprecedented speeds.

Insilico has entered into "a significant deal of up to **$2.75 billion**, including **$115 million** up front with pharma giant **Eli Lilly"** to rapidly develop new drugs, some intended to counteract **biological aging**.[18]

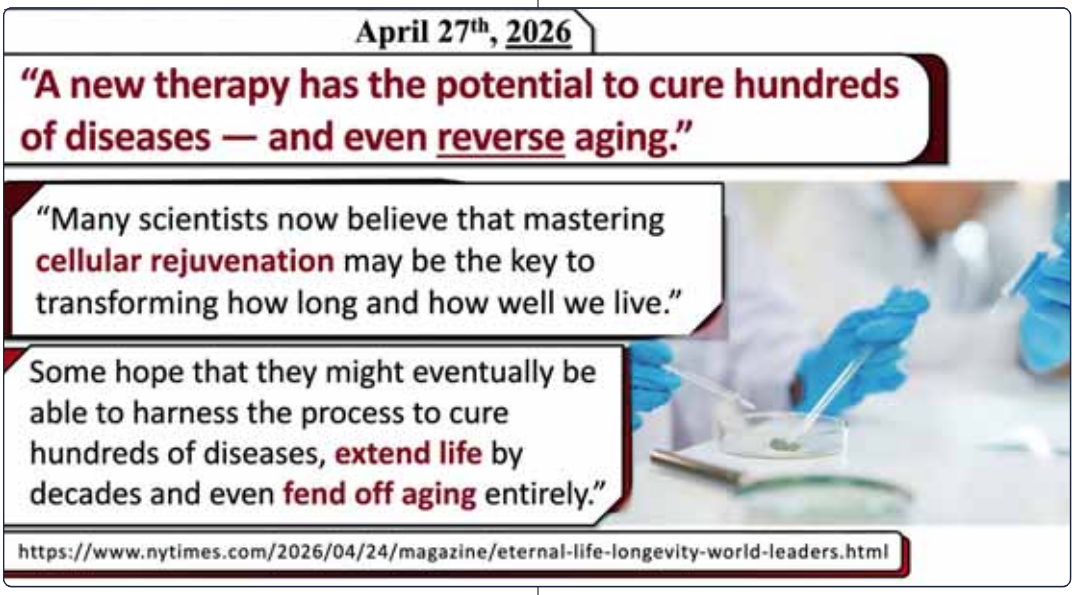
AI is compressing timelines that once required decades of pharmaceutical development.

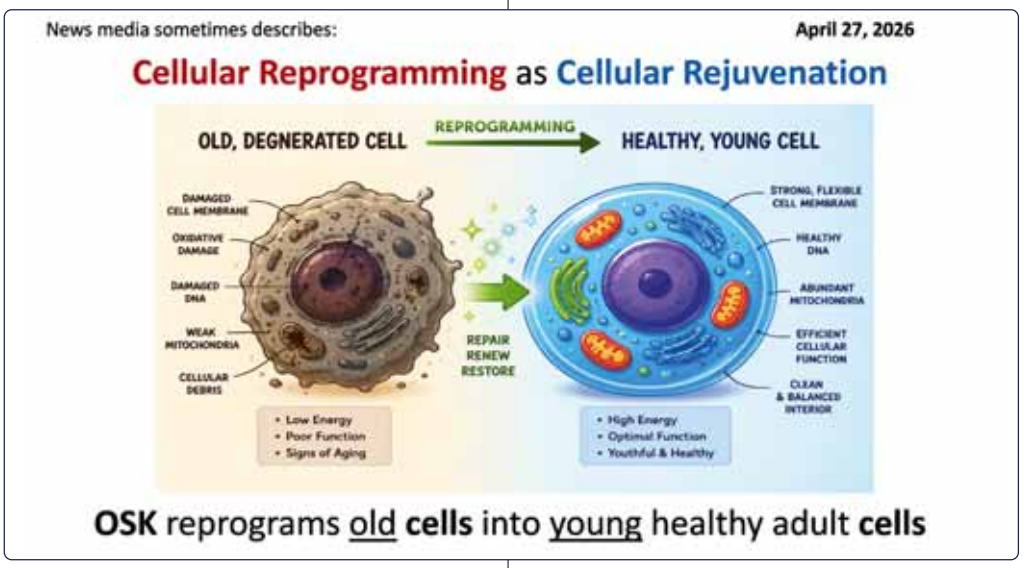
These computational systems can predict candidate drugs, optimize structures, and identify targets far faster than traditional approaches.[17]

The intersection of AI, genomics, and biologics is producing a beneficial compounding effect.

I view this as a "**biomedical renaissance**" where **aging research** is advancing exponentially.

To put this into historic perspective, prior to year **2014**, there was little in the way of **regenerative therapies** we could expect to become clinically available in the foreseeable future. Today multiple youth restoration **interventions** are being aggressively studied.





Rapid Fire Progress

Fibrosis is the excessive accumulation of dysfunctional connective tissue and other extracellular matrix components. This pathological process contributes to countless **deaths** worldwide.[19,20]

Think pulmonary **fibrosis**, cardiac **fibrosis**, liver **fibrosis**, kidney **fibrosis** et al.

Fibrosis and its inflammation-associated damage are implicated in **degenerative** aging.[21]

A recently identified mediator of **fibrosis** is a cytokine called **interleukin-11** (IL-11).[22]

A study published in 2024 showed that neutralizing IL-11 in mice improved healthspan and median lifespan **(23–25%)** when given to 75-week-old mice (roughly equivalent to 55 years old in **human** years).[23,24]

These findings are significant because they beneficially circumvent multiple **age-related** mechanisms. This includes measures of frailty, metabolism, fibrosis, and cancer incidence.

Research involving **IL-11 inhibitors** has drawn attention from biotechnology firms and longevity-focused investors.[23,25,26] **Calico** (Google's research arm) struck a deal to develop an IL-11–targeting drug, paying $25 million up front with up to $571 million in milestone payments ($600 million total).[27]

What's exciting is a potent **IL-11 inhibitor** drug advanced into clinical trials in **2025**[28] only **18 months** after the **mouse** study was published![23] It usually takes many years for research to move from **mouse** to **human** study…if ever.

If this **anti-IL-11 antibody drug** proves effective in humans, we may add it to our list of repurposed drugs (like **metformin**) that help neutralize pathologic aging processes.

Most of our readers supplement with nutrients with anti-fibrotic activity including **curcumin,**[29,30] **resveratrol**,[29] and **quercetin**.[29-31] If the anti-IL-11 antibody **drug** becomes available, I anticipate it will be more effective in suppressing the pathologic (IL-11) cytokine.

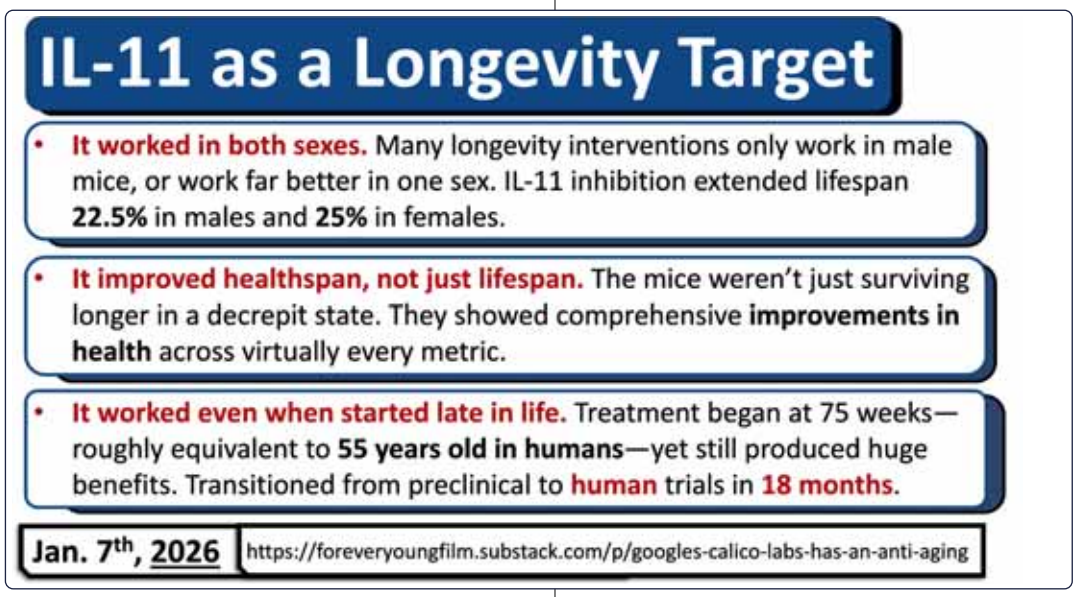
The fact that we are living in this era of rapid **innovation** is *beyond* fascinating. We may **live long** enough to personally benefit.

Huge Amounts of Research Funding

The media has extensively reported on the growing role of wealthy investors and major biotechnology firms entering the **longevity research** field.

An enormous influx of capital is pouring into charities and companies pursuing **rejuvenation science**. Recent advances have moved anti-aging research from fringe science into mainstream biotechnology.

Most groups involved in funding distinguish between extending lifespan and improving healthspan. They argue that the central goal is not merely increasing years lived, but preserving *youthful* function, cognition, mobility, and independence.



A Tidal Wave of Longevity Research

Aging is the dominant risk factor underlying most chronic diseases.[32] Rather than treating cancer, dementia, heart disease, and frailty as separate phenomena, increasing numbers of influential individuals recognize that most degenerative diseases are downstream consequences of **aging** biology.

And while improved healthspan is an often-stated objective, the benefit of increased **lifespan** is being recognized as a bridge to future advances in **medicine**.

In other words, individuals today may benefit by engaging in interventions to remain **alive** long enough to benefit from revolutionary rejuvenation therapies.

This is one of several motivating factors that is causing **billions** of dollars of mostly private money to go toward **longevity research**…enabling a virtual **tsunami** of discoveries/innovation.

Systemic Interventions

Successful **age reversal** will require **interventions** that have **system-wide** regenerative effects. This differs from single-pill studies that often do not yield meaningful results.

Laboratory and clinical advances today are improving gene regulation, inflammatory signaling, stem cell functionality, and regenerative capacities.

What was once viewed as speculative anti-aging enthusiasm now involves major universities, pharmaceutical companies, AI laboratories, and billions of dollars of investment capital.

The implications are profound. If biological aging can be slowed, halted, or partially reversed, then medicine can shift from treating individual diseases toward preserving ***youthful*** function itself.

The emergence of **rejuvenation biotechnology** may ultimately represent the largest transformation in human history — not simply adding years to life, but potentially redefining what aging means altogether.

My recurring messaging is urgency. We are all degenerating.

I use proceeds from Life Extension® **blood tests** and **supplement sales** to support several promising longevity projects.

Your ongoing patronage is enabling precedent setting advances in the biomedical research arena.

For longer life,

William Faloon, Co-Founder
Life Extension

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