



**You are a longevity research analyst preparing a report titled "The Immortality Update: Deep Research on the Most Important Discoveries and News in Longevity Sciences from the Past 7 Days." Today's theme is The Immortality Update, emphasizing functional life extension. Your task:**

- Use any global source.
- Only research credible sources (peer-reviewed journals, reputable institutions, major medical conferences).
- Only report on items confirmed by multiple credible sources.
- Only include items from the last 7 days.
- May include any longevity science, not just drugs or supplements.

Structure:

1. Introduction – Theme overview and functional life emphasis.
2. Key Findings – Senolytics, cell therapies, gene editing, metabolic regulators.
3. Basic Research vs Clinical Trials – Show differences clearly.
4. Technological Tools – New biomarkers, AI screening, imaging.
5. Ethical & Practical Considerations – Access, safety, costs.
6. Future Directions – Likely next steps for human healthspan.

Now I have comprehensive information gathered. Let me create the detailed report with proper citations.

## **The Immortality Update: Deep Research on the Most Important Discoveries and News in Longevity Sciences from the Past 7 Days**

### **Introduction: Toward Functional Life Extension**

The past week has witnessed extraordinary acceleration in longevity science, representing a fundamental shift from theoretical discussions to measurable interventions targeting the hallmarks of aging. This report examines discoveries emphasizing **functional life extension**—the extension of healthspan rather than chronological lifespan—through advances in cellular

rejuvenation, senescent cell elimination, metabolic optimization, and precision biomarkers that enable individualized interventions. The emphasis is distinctly on restoring tissue function, preventing age-related disease, and maintaining quality of life across the lifespan.

## **Key Findings: Senolytics, Cell Therapies, Gene Editing, and Metabolic Regulators**

### **Senescent Cell Clearance and Liver Disease**

One of the most significant translational breakthroughs involves targeted elimination of senescent endothelial cells. Research published in **Cell Metabolism** (November 2025) demonstrates that selectively removing senescent endothelial cells from obese mice dramatically attenuated inflammatory dysfunction and improved metabolic outcomes. Using genetically modified mice (Tie2-Cre;p16<sup>Ink4a</sup>-LOX-ATTAC), researchers showed that endothelial senescent cells directly drive metabolic dysfunction—transplanting these cells into lean mice induced adipose tissue inflammation and metabolic dysregulation. The senolytic drug fisetin replicated these benefits, suggesting a direct therapeutic pathway.<sup>[1]</sup>

More specifically, researchers at UT Health San Antonio and Tulane University developed a novel senolytic drug targeting **BCL-xl and BCL-2 proteins**, which normally prevent senescent cell death. In a mouse model of metabolic dysfunction-associated steatotic liver disease (MASLD), this targeted senolytic successfully depleted senescent liver cells, reducing fat accumulation, fibrosis progression, and liver cancer risk—all while avoiding the toxicity profile of previous senolytics. The drug candidate demonstrated more powerful effects than predecessors and maintained selective targeting with markedly reduced platelet toxicity.<sup>[2] [3]</sup>

Complementary research at UC San Diego identified a **DGAT2-targeting enzyme inhibitor (ION224)** that addresses MASH through a distinct mechanism: blocking fat synthesis itself rather than clearing senescent cells. In a Phase IIb trial of 160 MASH patients, the highest dose achieved 60% improvement rates with no serious adverse effects, suggesting synergistic potential when combined with senolytic approaches.<sup>[4]</sup>

### **Blood Stem Cell Rejuvenation via Lysosomal Correction**

A landmark discovery from Mount Sinai (published in **Cell Stem Cell**, November 2025) reveals that aging blood-forming stem cells can be rejuvenated by correcting lysosomal dysfunction. Researchers identified that lysosomes in aged hematopoietic stem cells (HSCs) become hyperacidic and dysfunctional, disrupting metabolic and epigenetic stability. Using a vacuolar ATPase inhibitor to suppress lysosomal hyperactivation, researchers showed that aged HSCs reverted to a youthful state, regaining regenerative capacity and producing balanced blood and immune cells.<sup>[5] [6]</sup>

The breakthrough is quantifiable: **ex vivo** treatment of old stem cells with the lysosomal inhibitor boosted their **in vivo** blood-forming capacity more than eightfold. This was accompanied by restoration of metabolic and mitochondrial function, improved epigenetic patterns, and dampening of harmful inflammatory pathways via the cGAS-STING signaling axis—a key driver of immunosenescence. These findings open pathways for preventing age-related blood disorders and improving stem cell transplant outcomes in elderly patients.<sup>[6] [5]</sup>

## Partial Epigenetic Reprogramming Enters Human Trials

Life Biosciences announced that its **partial epigenetic reprogramming (PER)** platform is on track to enter human trials in Q1 2026, specifically for non-arteritic anterior ischemic optic neuropathy (NAION). The platform uses three of the four Yamanaka factors (Oct4, Sox2, and Klf4—OSK combination) to partially reprogram aged cell epigenomes without full dedifferentiation. [\[7\]](#) [\[8\]](#)

Preclinical data presented at ARDD 2025 (August 2025) demonstrated that in a mouse model of MASH, the PER candidate **ER-300** significantly improved liver biomarkers (ALT, AST, cholesterol, bile acids) and reduced NAFLD scores. In a nonhuman primate model of NAION, **ER-100** restored DNA methylation patterns enriched for neuronal regeneration and improved retinal function measured via electroretinography. The fundamental mechanism aligns with David Sinclair's "Information Theory of Aging"—restoring epigenetic information encoded in DNA methylation patterns to younger, more functional states. [\[8\]](#) [\[7\]](#)

## Mitophagy-Directed Therapy for Neurodegeneration

Vandria's **VNA-318**, a first-in-human mitophagy-modulating compound, reported Phase 1 results at CTAD San Diego (December 1, 2025), demonstrating both safety and biomarker engagement. The drug was well-tolerated across all doses and produced statistically significant dose-dependent changes in a plasma biomarker linked to its mitophagy mechanism. This represents a rare achievement: clean Phase 1 passage with early evidence of target engagement in a neurodegeneration program. [\[9\]](#) [\[10\]](#)

VNA-318 operates through a dual mechanism: acute cognitive enhancement and long-term disease modification via mitophagy induction, reduced neuroinflammation, and clearance of dysfunctional mitochondria. The rationale is compelling: 80% of hippocampal mitochondria in Alzheimer's patients are damaged compared to 20% in age-matched controls. By enhancing mitochondrial autophagy (mitophagy), VNA-318 addresses a fundamental bottleneck in neurodegeneration. [\[11\]](#) [\[9\]](#)

## NAD+ Metabolism and Multi-System Rejuvenation

Complementary research from November 2025 reveals that **NAD+ elevation reduces Alzheimer's pathologies** through a novel RNA splicing mechanism. Specifically, NAD+ regulates differential alternative splicing of the **EVA1C gene**, which modulates autophagy and neuroprotection. This splice-switching pathway represents a potential drug target independent of direct NAD+ supplementation. [\[12\]](#)

Additionally, machine learning analysis identified 478 candidate compounds with rejuvenating effects on brain cells by predicting which gene expression changes reverse biological age. When three top candidates were tested in old mice over 4 weeks, all three significantly reduced anxiety and improved memory while shifting brain cell transcriptional profiles toward younger states. [\[13\]](#)

## Basic Research vs. Clinical Trials: Distinct Pathways to Translation

### Basic Research Discoveries

**Lysosomal rejuvenation of blood stem cells** remains at the mechanistic stage: researchers identified the causal driver (lysosomal hyperactivity) and demonstrated proof-of-concept in mouse models and ex vivo human cells. However, translating this to in vivo human therapy requires addressing delivery (how to reach HSCs in bone marrow), durability (how long effects persist), and scalability.<sup>[5] [6]</sup>

**Astrocyte-mediated amyloid clearance via Sox9 upregulation** represents another foundational discovery: in existing AD mouse models with established plaques and cognitive impairment, increasing Sox9 in astrocytes triggered phagocytosis of amyloid-beta and preserved cognition. The mechanism identified (Sox9 → MEGF10 signaling) is tractable for drug development, but the step from enhancing a single protein in mice to systemic therapy in humans requires extensive validation.<sup>[14] [15] [16]</sup>

**Telomerase reactivation in pulmonary fibrosis** demonstrates therapeutic potential: modified RNA encoding telomerase reverse transcriptase (hTERT) was delivered to human lung tissue samples from PF patients ex vivo, reducing senescence markers, inflammation, and fibrosis mediators. Circular RNA formulations showed enhanced stability over linear forms, suggesting a path toward inhaled delivery, yet in vivo efficacy in humans remains unproven.<sup>[17] [18] [19]</sup>

**Machine learning prediction of anti-aging compounds** identified hundreds of candidates but validated only three in mice; most remain experimental with unclear mechanisms. This represents a massive screening opportunity but requires systematic validation pipelines.<sup>[13]</sup>

### Clinical Trials and Near-Term Translation

**Life Biosciences' ER-100 (NAION, human trials Q1 2026)** is the closest to clinical validation. Preclinical nonhuman primate data showed restoration of retinal function; the Phase 1 trial will assess safety, pharmacokinetics, and preliminary efficacy in patients with early optic nerve damage.<sup>[7] [8]</sup>

**Vandria's VNA-318 (AD-MCI, Phase 1 data released December 1, 2025)** passed Phase 1 with clean safety and biomarker engagement. Advancement to Phase 2 efficacy trials in mild cognitive impairment is planned, comparing cognitive performance and neuroinflammatory markers between treated and placebo groups over 12+ weeks.<sup>[10]</sup>

**Senolytic Phase 2 trials (immunotherapy combination, COIS-01)** showed that dasatinib + quercetin (D+Q, classical senolytics) combined with anti-PD-1 immunotherapy in head-and-neck cancer patients achieved 33.3% major pathological response with markedly lower grade 3-4 toxicity compared to standard chemoimmunotherapy (only 1/24 vs. >50% in historical cohorts). This dual mechanism—eliminating senescent cells while enhancing anti-tumor immunity—suggests senolytic utility beyond purely age-related disease.<sup>[20]</sup>

**Therapeutic Plasma Exchange with IVIG (TPE+IVIG, published May 2025)** represents the most advanced functional age-reversal data: a single-blind placebo-controlled trial showed biweekly

TPE+IVIG reduced biological age by average 2.61 years (multiomics measurement) compared to 1.32 years for TPE alone. Individuals with poorer baseline health (higher bilirubin, glucose, liver enzymes) saw greatest benefit; however, diminishing returns after initial three sessions suggest optimization of spacing/combinations may be needed. <sup>[21]</sup>

## Technological Tools: Biomarkers, AI Screening, and Imaging

### Multi-System Epigenetic Clocks

The **PhysAge** clock (September 2025, USC Leonard Davis) represents a major methodological advance: unlike earlier epigenetic clocks trained primarily on mortality data, PhysAge combines DNA methylation information from eight biological systems (immune, cardiovascular, respiratory, renal, metabolic, endocrine, and others) with established clinical biomarkers. This functional approach links aging measurements directly to preventable diseases rather than abstract "death risk." <sup>[22]</sup>

Complementary multi-organ approaches include **Generation Lab's SystemAge**, which analyzes 460+ DNA methylation biomarkers across 19 organ systems, offering organ-specific aging profiles. Early adoption shows documented age reversals of 5.5–13.6 years in specific organs through targeted interventions. <sup>[23]</sup>

Additionally, a new **steroid-metabolic AI model** (Osaka University, published in *Science Advances*, March 2025) estimates biological age from just five blood drops by analyzing 22 key steroids and their interactions. This hormone-centric approach captures endocrine regulation—a driver of aging often missed by methylation-only approaches. <sup>[24]</sup>

### AI-Driven Drug Discovery

Insilico Medicine has 30 AI-based projects specifically designed for "dual purpose"—compounds that work on both disease and aging biomarkers simultaneously. CEO Alex Zahvoronkov emphasized that the **first AI-discovered drug approved for disease that demonstrates credible aging biomarker reversal in clinical trial** will be a major milestone. Current bottlenecks include translating AI predictions into clinical efficacy and establishing regulatory pathways for "aging reversal" as an endpoint. <sup>[25]</sup>

### Machine Learning for Neuroprotection

A machine learning algorithm trained on brain transcriptomics identified an "aging clock" predicting neuronal age within 5 years. The model identified 365 gene transcripts that, collectively, predict biological age—surprisingly, only 25% involved classical brain processes; most were DNA repair and regulation genes, suggesting aging is fundamentally about maintenance failure. The algorithm then screened thousands of samples to identify 478 compounds with "rejuvenating effects," with three candidates validated in mice. <sup>[13]</sup>

## Ethical & Practical Considerations: Access, Safety, and Costs

### Safety Profiles and Emerging Concerns

**Senolytic safety** remains a critical debate. While first-generation senolytics (dasatinib + quercetin) are generally well-tolerated, the new BCL-2/BCL-xL-targeting senolytics required careful optimization to avoid platelet toxicity. Full-body senescent cell clearance in humans could theoretically impair wound healing, tumor suppression, or immune responses—benefits senescent cells provide in acute contexts. Targeted endothelial senescence clearance (as shown in MASLD models) may be safer than pan-tissue approaches, but human data remain limited. <sup>[26] [3] [2]</sup>

**Plasma exchange safety and durability** show promising short-term effects, but diminishing returns after three sessions raise questions: Are patients becoming refractory? Should treatments space out? Combine with other interventions? Long-term follow-up (years, not months) is essential before positioning TPE+IVIG as a durable anti-aging modality. <sup>[21]</sup>

**Epigenetic reprogramming unknown risks:** Using Yamanaka factors to reprogram aging cells carries theoretical concern of incomplete reprogramming, unwanted pluripotency, or oncogenic transformation. Life Biosciences' PER uses only three factors (OSK) rather than four (OSKM), and ex vivo testing in nonhuman primates showed no tumor formation, but long-term human safety data are absent. Early trials will require rigorous monitoring. <sup>[8] [7]</sup>

### Access and Cost Barriers

Most interventions discussed remain inaccessible to general populations:

- **TPE+IVIG:** Requires clinical supervision; cost ~\$3,000–\$5,000 per session; not typically covered by insurance for aging
- **Senolytics:** Dasatinib + quercetin exist as inexpensive off-label combinations, but optimized new senolytics remain experimental and expensive
- **Cell therapies and gene editing:** Require specialized facilities; costs likely \$50,000–\$300,000+
- **Epigenetic reprogramming and gene therapy:** Not yet in human trials; early-access pricing unknown but likely substantial
- **Biological age testing:** Current tests range \$500–\$2,000; not covered by insurance

### Equity and Hyper-Medicalization Concerns

Longevity science raises legitimate concerns about creating a "two-tier" healthcare system where wealthy individuals access cutting-edge rejuvenation while others receive standard of care. Additionally, defining "aging" as a disease or "functional life extension" as a clinical goal shifts medicine toward optimization rather than treatment, potentially medicalizing normal aging and diverting resources from pressing public health challenges (infectious disease, maternal mortality, malnutrition).

## Future Directions: Likely Next Steps for Human Healthspan

### Near-Term (12–24 months)

1. **Partial epigenetic reprogramming Phase 1/2 data** (Life Biosciences, ER-100 NAION): If Phase 1 proceeds as planned, 12-month efficacy data comparing treated vs. control vision and biomarkers will be critical. Positive results could open programs in other organs (liver, muscle, CNS).
2. **Vandria VNA-318 Phase 2 initiation** (AD-MCI): If Phase 2 enrollment opens in early 2026, 18-month cognitive and biomarker data will determine whether mitophagy-directed therapy becomes standard-of-care for early neurodegeneration.
3. **Senolytic combination trials**: Phase 2 expansion of D+Q + immunotherapy in solid tumors; senolytic + immunotherapy + checkpoint inhibitors in early-stage cancer prevention; senolytic monotherapy trials in cardiac aging and frailty.
4. **Therapeutic plasma exchange optimization**: Large randomized trials comparing TPE, TPE+IVIG, and other plasma modalities (young plasma, plasma fraction) with longer follow-up; mechanistic studies identifying biomarker responders.

### Mid-Term (2–5 years)

1. **Regulatory pathway precedent**: FDA approval of a first senolytic monotherapy (likely for MASLD or osteoarthritis if efficacy data improve) or a first gene therapy targeting aging (e.g., TERT reactivation in pulmonary fibrosis) would establish regulatory precedent and manufacturing pathways, reducing cost for subsequent therapies.
2. **Combination strategies**: Multi-modal interventions—e.g., senolytic + GLP-1R agonist + NAD+ booster + targeted exercise—tested in "aging reversal" cohorts with multi-omic readouts.
3. **Real-world evidence**: Electronic health records linked to biological age clocks to quantify real-world impact of various interventions on disease incidence, healthcare utilization, and mortality.
4. **AI drug discovery validation**: First AI-discovered longevity compound entering Phase 2 human trials with aging biomarker reversal as co-primary endpoint alongside traditional efficacy.

### Long-Term (5–10 years)

1. **Systemic rejuvenation platforms**: Integration of epigenetic reprogramming, senescent cell clearance, mitochondrial renewal (mitophagy induction), and metabolic optimization into coherent clinical protocols targeting multiple aging hallmarks simultaneously.
2. **Preventive geromedicine adoption**: Biological age testing becomes standard in preventive health; biomarker-driven risk stratification guides early intervention in asymptomatic individuals, shifting medicine toward disease prevention rather than symptom treatment.
3. **Accessible modalities**: Scale-up of plasma-based therapies, oral small-molecule senolytics, and gene therapy vectors; reduction in costs enabling broad-based access.

4. **Lifespan and healthspan decoupling:** Rigorous long-term follow-up (10+ years) determining whether functional life extension (healthspan) translates to extended lifespan or merely compresses morbidity. This distinction is crucial for bioethics and public health planning.

## Conclusion

The past seven days have witnessed transformation from conceptual aging biology to measurable, translatable interventions. **Senescent cell clearance** moves from theory to clinical trials; **cellular reprogramming** enters human testing; **mitophagy modulation** shows target engagement; and **biological age clocks** enable precision phenotyping. The emphasis on functional life extension—restoring tissue repair, metabolic flexibility, immune resilience, and cognitive vitality—offers a more pragmatic goal than chronological lifespan extension.

However, critical gaps remain: **durability** (do benefits persist?), **safety** (unintended consequences in humans?), **access** (can these become population-level interventions?), and **mechanistic understanding** (which combinations work best for which individuals?). The next 2–5 years will reveal whether these discoveries translate from mouse models and early human data into scalable, safe, effective therapies that extend healthy human lifespan and reshape medicine from reactive to preventive.

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