The Immortality Update: Longevity Science Discoveries from November 18-25, 2025

The past week delivered breakthrough biological age clocks, stem cell vision restoration in humans, and evidence that common diabetes drugs may combat aging body-wide—while funding uncertainties cast shadows over the field's momentum.

This week's developments converge on a powerful theme: aging is becoming measurable and increasingly modifiable. Two landmark AI-powered biological clocks published in Nature journals now enable aging assessment from smartwatches and routine medical records. Meanwhile, clinical and preclinical advances revealed new therapeutic targets—from lysosomes in blood stem cells to hydrogen sulfide regulation via sirtuins—that could extend healthy function rather than merely add years. These discoveries matter because they're shifting longevity science from theoretical biology toward practical medicine, even as a major industry partnership collapse and proposed NIH funding cuts remind us that progress remains fragile.

Stem cell therapy restores vision in age-related blindness

The week's most clinically advanced finding came from the University of Michigan, where a Phase 1/2a trial demonstrated that transplanted adult retinal stem cells can restore sight in patients with advanced dry age-related macular degeneration (AMD). ScienceDaily Published November 22 in *Cell Stem Cell*, the trial treated six participants with **50,000 adult-derived retinal pigment epithelial cells** during eye surgery. sciencedaily

Results after one year were remarkable: participants gained the ability to read an average of **21 additional letters on eye charts**—unprecedented for this severely affected population. Crucially, the treatment showed no serious inflammation or tumor growth. sciencedaily Twelve additional participants are now being monitored at higher doses (150,000 and 250,000 cells). ScienceDaily sciencedaily This represents one of the first restorative treatments for dry AMD, which affects over 20 million Americans and currently lacks any vision-restoring therapy.

Separately, Mount Sinai researchers announced on November 24 that they successfully reversed aging in blood-forming stem cells by targeting lysosomal dysfunction. Their *Cell Stem Cell* study found that lysosomes in aged hematopoietic stem cells become hyper-acidic and abnormally activated. By suppressing this hyperactivation with a vacuolar ATPase inhibitor, they restored old stem cells to **more than eight-fold improved blood-forming capacity**, including balanced immune cell production, renewed mitochondrial function, and reduced inflammatory signaling. mountsinai This preclinical work opens therapeutic avenues for age-related blood disorders and improving stem cell transplants in older patients. Mount Sinai

Six genes emerge as causal aging drivers

A cross-species computational analysis published November 18 identified six specific genes that causally drive aging—not merely correlate with it. Researchers at Washington University School of Medicine pooled 25 geneexpression datasets from humans, dogs, and rodents, then validated findings by silencing corresponding genes in *C. elegans*. Longevity.Technology Inhibiting any of these six genes extended worm lifespan by **8-15%**. longevity

The genes fall into two categories: those that increase with age (CASP1, an inflammasome protease; RSRC1, involved in RNA splicing) and those that decrease (CA4, a carbonic anhydrase; SPARC, an extracellular matrix protein; CDC20, cell division; DIRC2, lysosomal transport). Longevity.Technology Importantly, CASP1 blockers are already under investigation for Alzheimer's disease, while CA4 is targeted by acetazolamide, a glaucoma medication that has extended lifespan in progeroid mouse models— longevity suggesting near-term translational potential.

A complementary November 21 study in *PLOS Biology* from Harvard and Baylor revealed that RNA splicing factors REPO-1 and SFA-1 determine whether longevity interventions work at all. Early-life splicing activity establishes a "cellular landscape" that enables responsiveness to later anti-aging treatments. PLOS plos Disrupting these factors blocked the benefits of dietary restriction and mTOR inhibition, plos suggesting that **individual response variation to anti-aging drugs may be predictable** through splicing biomarkers—a step toward personalized geroscience.

GLP-1 drugs show body-wide rejuvenation effects in mice

Perhaps the week's most consequential finding for near-term clinical application came from Chinese University of Hong Kong, published November 19 in *Cell Metabolism*. Researchers treated middle-aged mice with low-dose exenatide (a GLP-1 receptor agonist in the same class as semaglutide/Ozempic) for 30 weeks and performed comprehensive molecular profiling across brain, liver, kidney, muscle, and fat tissue.

The results showed **strong body-wide age-counteracting effects** including improved grip strength, rotarod performance, and cognition—crucially, at doses that minimally affected food intake or body weight, and specifically in aged mice rather than young adults. Cell Press Brain activity appeared to serve as a hub influencing aging profiles across organs. bioRxiv Combined with earlier 2025 data showing semaglutide reduces epigenetic age markers (GrimAge) by 1.4-2.3 years, this provides mechanistic evidence that widely-prescribed metabolic drugs may have broad anti-aging effects. Eli Lilly is reportedly planning a TAME-like study with a GLP-1 agonist.

Also on November 18, Bar-Ilan University researchers published in *PNAS* how the longevity protein Sirt6 precisely regulates hydrogen sulfide (H_2S) production—a gasotransmitter supporting wound healing, heart health, and brain function that declines with age. Unlike calorie restriction, which broadly raises H_2S, Sirt6 acts with precision to maintain optimal levels while preventing harmful overproduction. EurekAlert! This identifies Sirt6 as a potential drug target for mimicking calorie restriction's benefits. Sentinelassam

AI-powered biological clocks reach new precision and accessibility

Two landmark biological age clocks debuted November 19 in Nature journals, marking a shift toward accessible, continuous aging measurement.

LifeClock, published in *Nature Medicine*, predicts biological age across all life stages using routine electronic health records. Nature Trained on over **24 million clinical visits from 9.7 million individuals**, Nature it uses

184 clinical indicators (nature) and achieves a mean absolute error of 4.14 years when validated against UK Biobank data. Beyond age prediction, it forecasts diseases including diabetes (AUC=0.98), coronary artery disease (AUC=0.98), and atrial fibrillation (AUC=0.95) years before onset. Uniquely, LifeClock includes a pediatric model distinguishing developmental processes from damage-based aging. (nature) (Nature)

PpgAge, published in *Nature Communications*, derives biological age from Apple Watch photoplethysmography signals—passively collected wrist-based cardiovascular data. Analyzing over **149 million participant-days** from 213,593 Apple Heart & Movement Study participants, it achieves a mean absolute error of just **2.43 years** in healthy individuals. (nature) A six-year PpgAge gap correlated with 3.5× higher heart disease rates and 2.4× higher diabetes rates. (nature) The clock detected real-time aging changes around cardiac events and even pregnancy (median 3.56-year increase during gestation). (nature)

Additionally, IGC Pharma announced November 25 its MINT-AD platform using AI to identify socioeconomic risk factors for Alzheimer's across 14 longitudinal aging studies spanning the US, Africa, Asia, Europe, and Latin America— (PharmiWeb) highlighting the global scope of modern aging research tools.

Basic research versus clinical trials at a glance

The week's findings span the research continuum from fundamental discovery to human treatment:

- **Human clinical trials**: AMD stem cell therapy (Phase 1/2a, Michigan), vision improvement documented
- **Preclinical animal studies**: Blood stem cell rejuvenation (Mount Sinai, mice), GLP-1 aging effects (Hong Kong, mice), Sirt6/H_2S mechanism (Bar-Ilan, mice), six-gene validation (Washington University, C. elegans), splicing factors (Harvard/Baylor, C. elegans)
- **Technology development**: LifeClock and PpgAge biological clocks (large-scale human validation studies, research stage)
- **Ongoing clinical trials**: PEARL rapamycin trial completed April 2025 showed safety in humans; TAME metformin trial remains unfunded; (Longevity.Technology) Loyal's dog longevity drug LOY-002 in pivotal study (DVM360) with 1,300+ dogs

The pattern reveals a field where mechanistic understanding is rapidly advancing through animal models, while human trials remain concentrated in narrow indications (AMD, metabolic disease) rather than aging per se.

Funding uncertainties and industry setbacks threaten momentum

The week also brought sobering reminders of longevity research's fragility. AbbVie announced November 17 it will end its 11-year, **billion-dollar partnership** with Alphabet-backed Calico Life Sciences, laying off approximately 100 scientists. (Longevity.Technology) (Statnews) The decision followed Calico's ALS drug fosigotifator failing clinical endpoints, though Calico has since licensed an IL-11 antibody program from China, (Longevity.Technology) suggesting refined rather than abandoned ambitions.

More broadly, proposed FY 2026 NIH budget cuts of approximately **40%** have created what researchers describe as "corrosive" uncertainty. (Longevity.Technology) Andrew Steele of The Longevity Initiative noted this would reduce federal longevity support "from $1.04 per American to 62 cents." While Congress has shown limited appetite for such dramatic cuts, the mere prospect reshapes institutional decisions—delaying hiring, discouraging early-career researchers, longevity and threatening multi-year cohort studies essential to aging research. Longevity.Technology

Buck Institute's Professor David Furman highlighted another challenge NutraIngredients.com on November 25: the "Hawthorne effect," where study participants improve health behaviors simply from being observed, producing 5-10% improvements that obscure treatment effects. He advocated citizen science approaches with tens of thousands of participants using real-world data to overcome this limitation.

Ethical dimensions demand attention as the field matures

Several ethical and access issues crystallized this week. **Cost barriers** remain severe: longevity clinic programs range from $15,000 to $250,000 annually, while GLP-1 drugs cost several hundred dollars monthly with inconsistent insurance coverage. Medicare doesn't cover GLP-1s for weight loss, raising concerns about a "longevity divide" where extended healthspan becomes a privilege of wealth.

The **international research landscape** is shifting notably. China's longevity program is attracting billion-dollar investment, with companies like Lonvi Biosciences developing grapeseed-derived anti-aging compounds. Harvard's Vadim Gladyshev observed that Chinese researchers "are rapidly catching up" Business Standard precisely as US funding faces uncertainty.

The Biomarkers of Aging Conference summary released November 24 noted increasing clinician presence longevity and regulatory discussions about frameworks for clinical deployment— Longevity.Technology signals that the field is maturing toward practical application, though questions of access, safety monitoring, and appropriate claims remain unresolved.

What comes next for human healthspan extension

This week's findings collectively suggest several near-term trajectories. **GLP-1 agonists** represent the most likely candidates for repurposing as longevity drugs, with existing safety data, widespread availability, and accumulating evidence of age-counteracting effects. Expect clinical trials specifically examining aging endpoints within 1-2 years.

Biological age clocks are transitioning from research curiosities to clinical tools. ScienceDirect LifeClock and PpgAge demonstrate that continuous, non-invasive aging measurement is achievable—enabling both individual monitoring and surrogate endpoints for drug trials. The Biomarkers of Aging Conference highlighted growing industry and clinician confidence in these tools.

Stem cell therapies are advancing condition by condition. AMD may be joined by optic neuropathy (Life Biosciences' partial reprogramming therapy targeting 2025-2026 trials) P05 as early proving grounds for regenerative approaches to aging. Longevity.Technology Labiotech.eu

The **six causal aging genes** and **Sirt6/H_2S pathway** discoveries offer new drug targets, with some compounds (acetazolamide, CASP1 inhibitors) already in clinical use for other conditions— longevity accelerating potential repurposing timelines.

However, the TAME trial's continued funding struggles (Fight Aging!) and AbbVie-Calico's collapse underscore that scientific possibility alone doesn't guarantee therapeutic translation. The path from mechanism to medicine in longevity remains long, expensive, and institutionally precarious.

Conclusion

This week established that aging's biological clock can now be read from a smartwatch and that stem cells can restore lost vision in aging eyes—two developments that would have seemed distant just years ago. The convergence of AI-powered measurement tools, GLP-1 drug repurposing evidence, and fundamental discoveries in lysosomal function, sirtuin biology, and genetic causation creates unprecedented momentum toward functional life extension. Yet the AbbVie-Calico dissolution and NIH funding uncertainty remind us that longevity science operates at the intersection of biological possibility and institutional will. The coming months will reveal whether this week's discoveries accelerate toward clinical reality or remain promising findings awaiting resources and regulatory pathways that may not materialize.