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

Introduction

Today's **Immortality Update** highlights the latest breakthroughs in longevity science, with an emphasis on interventions aimed at extending **functional lifespan** – keeping us healthier and more capable as we age, not merely prolonging years. In the past week, researchers and innovators worldwide have announced findings that bring us closer to **extending healthspan**, from cellular therapies and gene-targeted drugs to novel biomarkers and AI tools. This report summarizes the high-impact discoveries of the last 7 days, explaining how they might enhance not just lifespan but the quality of life in our later years. We'll distinguish early-stage lab results from clinical trial milestones, showcase cutting-edge technologies accelerating progress, and consider the ethical and practical implications of pursuing longer, healthier lives.

Key Findings

• **A next-generation TOR inhibitor extends lifespan via a new metabolic mechanism:** Scientists unveiled *Rapalink-1*, a new drug that can mimic and enhance the anti-aging effects of rapamycin [1]. In yeast experiments, Rapalink-1 significantly **extended lifespan** comparable to rapamycin, while **upregulating enzymes** (agmatinases) that break down the gut metabolite **agmatine** [1]. This revealed a **previously unknown feedback loop** in metabolism: by activating agmatinase enzymes, Rapalink-1 keeps the pro-aging TOR pathway in check [2]. The discovery is important because it links **microbiome-derived compounds** to longevity pathways. Researchers suggest this mechanism may be **conserved in humans**, offering clues to how nutrition and gut bacteria influence aging [3] [4]. Rapalink-1 works through the TORC1 growth pathway, essentially fooling cells into a "low nutrition" state that slows growth but **prolongs cell survival** [5] [6]. While human applications are years away, this finding provides a clearer map of TOR signaling and metabolism that could be targeted to **delay aging and age-related diseases** [7].

Caenorhabditis elegans, the tiny roundworm, is a powerful model for aging studies. Researchers found that altering just two glial cells in the worm's nervous system can trigger protective changes throughout the body. [8] [9]

• **"Alkalizing" brain support cells boosts lifespan and neuroprotection (in worms):** A team at the University of Miami discovered that tweaking the internal chemistry of **glial cells** can extend healthy life. In *C. elegans* worms, deleting a chloride channel gene (**CLH-1**) in just **two glial cells** led to longer lifespan and sustained mobility with age [9]. The treated worms showed **enhanced stress resilience**, with lower oxidative damage and higher **autophagy** (cellular cleanup), and even resisted protein aggregates in a Huntington's disease model [10] [11]. Mechanistically, the loss of CLH-1 made the glial cells more **alkaline** (higher pH), which turned on a cascade of anti-aging genes [12]. If researchers neutralized this alkalinity, the benefits disappeared, confirming that a **more alkaline glial environment** was key [12]. Conversely,

artificially raising glial cell pH in normal worms replicated the longevity and neuroprotective effects [12]. This novel finding positions **glial cells** – often overlooked next to neurons – as **master regulators of aging**. It suggests that modulating brain cell pH or ion channels might one day become a strategy to promote **healthy aging and combat neurodegeneration** [8] [13]. The authors note that mammals share these fundamental processes, raising hopes that a similar approach could work in mice or humans [14].

• **Protecting the blood-vessel lining prevents frailty in aging (mouse study):** New research highlights the importance of the **glycocalyx**, a microscopic gel-like layer lining our blood vessels, in maintaining muscle health during aging. In a paper published in *Aging-US*, scientists showed that preserving the glycocalyx can **prevent age-related muscle loss and performance decline** [15]. Older mice fed a diet enriched with **high-molecular-weight hyaluronan (HMW-HA)** – a major glycocalyx component – for 10 weeks retained their muscle mass and **exercise capacity** compared to untreated aged mice [16] [17]. Treated mice ran longer on treadmills and did not exhibit the usual losses in strength and endurance that accompany aging [17]. In contrast, untreated control mice of the same age lost muscle volume and strength over the study period [17]. The team also studied genetically modified mice lacking **Has2** (an enzyme required for HMW-HA production) and found that a thinned glycocalyx led to impaired exercise performance and muscle mitochondrial function – even without muscle atrophy [18] [19]. This indicates that **glycocalyx degradation directly contributes to frailty**, likely by hampering blood flow and nutrient delivery to muscles [20] [21]. The exciting implication is that **glycocalyx-targeted therapies** (like the HMW-HA supplement, known as Endocalyx™) might combat frailty and mobility loss in the elderly [16] [22]. While these results are in animals, they provide a strong rationale to explore similar vascular-protective strategies in humans to **extend healthspan**.

• **Senolytic and stem cell therapy proposed to "re-awaken" chronic coma patients:** In a bold intersection of longevity tech and acute medicine, a longevity biotech company (Immorta Bio) published a peer-reviewed paper outlining a novel two-step approach to restore consciousness in patients with severe brain injury (coma). The strategy combines **senolytic immunotherapy** – using their *SenoVax*™ platform to clear out toxic senescent cells and reduce inflammation in the brain – with an infusion of the patient's own **mesenchymal stem cells** to spur neural repair [23] [24]. The idea is that aging or injury leaves behind senescent cells that **promote inflammation and hinder recovery**; by removing them, the brain's environment becomes more conducive to healing [25] [26]. The autologous (self-derived) stem cells, delivered alongside, could then more effectively regenerate damaged neural circuits [27] [28]. **Preclinical studies** cited in the paper support that clearing senescent cells can reduce neuroinflammation and that stem cells can aid neural recovery [29] [28]. Intriguingly, the authors report **preliminary cases** where patients showed restored responsiveness and cognitive function after such combined treatment [28]. If validated, this approach would mark a paradigm shift – treating coma not as an untreatable state but as a condition of aging/injury that can be reversed by **rejuvenating the cellular environment and stimulating repair** [30]. Beyond coma, this research hints at wider applications: the senolytic+stem cell synergy might also be tested in neurodegenerative diseases like Alzheimer's and Parkinson's, where senescent cells and cell loss both play roles [30]. It's a striking example of longevity science (targeting cellular senescence) being applied to improve functional recovery in a critical medical condition.

• **First mitochondria-targeted drug wins FDA approval, hinting at broader longevity benefits:** In regulatory news, **Stealth BioTherapeutics'** drug *Forzinity* (elamipretide) received FDA accelerated approval – a milestone as the **first therapy targeting mitochondrial dysfunction** to reach the market [31]. The drug was approved to treat **Barth syndrome**, a rare genetic disease in which faulty mitochondria lead to life-threatening heart failure and muscle weakness in young males [32] [33]. Forzinity works by binding to the

inner mitochondrial membrane, **improving mitochondrial structure and function** [33]. In clinical trials, it improved patients' leg muscle strength (the muscle used to extend the knee), an outcome the FDA deemed likely to predict real functional gains (such as standing or walking more easily) [33] [34]. This approval is significant beyond the rare disease: it is the **first formal acknowledgment by regulators that targeting mitochondrial health can translate into human therapeutic benefits** [35]. Mitochondrial decline is implicated in many age-related conditions (from neurodegeneration to frailty), so this success "validates the scientific and clinical rationale" behind a host of longevity biotech efforts aimed at the mitochondria [31] [36]. Longevity experts note that mitochondrial membrane stability correlates with species' lifespans, and drugs like elamipretide (Forzinity) which **stabilize mitochondria** could hold promise for treating common diseases of aging [37]. Indeed, Stealth is already testing related compounds in age-linked conditions like dry macular degeneration [38]. By **improving cellular energy production**, such therapies might not only treat specific diseases but also **"restore function and improve healthspan,"** as Stealth's CEO remarked [39]. The ripple effect of this approval is expected to accelerate investment in similar approaches and pave the way for future "geroprotective" drugs that target fundamental aging processes to keep people healthier longer [36] [37].

Early-Stage Research vs. Clinical Trials

Recent longevity findings run the gamut from **basic science breakthroughs** in model organisms to **clinical advances** in humans. It's important to distinguish where each stands on the path to real-world application:

- **Laboratory and Animal Studies:** Many headline-grabbing results this week are in early models – yeast, worms, or mice – offering mechanistic insights but not yet ready for human use. For example, the Rapalink-1 study was done in yeast cells, and while it uncovered a compelling longevity mechanism, any potential "anti-aging pill" is far off [7]. Similarly, the glial-cell alkalinity discovery in worms and the glycocalyx-focused muscle aging study in mice demonstrate *proof-of-concept* that tweaking certain pathways can extend healthspan in those models [40]. These **preclinical** findings guide our understanding of aging biology and identify targets (e.g. TOR signaling, glial pH, vascular glycocalyx) for intervention. However, effects in simple organisms or rodents don't guarantee the same in humans, and safety is a major unknown at this stage. Researchers stress that more work – often years of additional animal studies – is needed before translating these ideas into therapies [40]. Caution is warranted; for instance, a supplement like agmatine showed longevity links in yeast, but the scientists advise against humans taking it for anti-aging based on current data [41].

- **Human Trials and Therapies:** In contrast, a few developments have progressed to or emerged from human trials, marking tangible steps toward clinical use. The Forzinity mitochondrial drug is a prime example of a longevity-related intervention proving itself in humans and gaining regulatory approval. Its clinical trial demonstrated functional improvement (muscle strength) in patients, which led the FDA to authorize its use [33]. This kind of evidence – actual health benefits in people – differentiates it from hypotheses tested only in labs. Another semi-clinical example is the coma reversal approach: while mostly theoretical, it is grounded in peer-reviewed science and there are indications (from case studies or initial trials) of patient responses [28]. Still, it remains **experimental** until validated in controlled clinical trials. Overall, we are seeing the first therapies (like Forzinity) emerging from longevity science enter medical practice, while many other strategies linger at the exploratory stage. The challenge ahead is to **bridge this gap** – to take promising strategies (senolytics, metabolic regulators, etc.) from bench to bedside through rigorous trials demonstrating not just longer life, but preserved **function and quality of life** for patients.

Technological Tools Accelerating Longevity Research

A striking theme in the past week's news is the role of **advanced technologies** – from AI to new biomarkers – in propelling longevity research forward:

- **AI-Driven Discovery and Analysis:** Researchers are increasingly leveraging artificial intelligence to decode aging and speed up breakthroughs. One notable development is the use of multi-agent AI platforms to dramatically **compress research timelines**. For example, a collaboration out of Harvard used an AI system called K-Dense to analyze huge transcriptomic datasets and build a new biological aging clock in a matter of weeks, a task that normally might take scientists months or years [42] [43]. Such AI platforms can design experiments, sift through omics data, and even draft reports, effectively acting like a team of digital researchers working around the clock [42]. Likewise, biotech companies are deploying AI in drug discovery: **Rubedo's ALEMBIC platform** used machine learning to identify a novel senotherapeutic compound (a GPX4 modulator that forces senescent cells to self-destruct) and design it in silico [44]. These AI tools can find patterns and targets in the **tsunami of biomedical data** that would overwhelm human analysis, from genetic pathways to structural biology. The past week also saw the launch of specialized longevity AI infrastructures – for instance, Elivion.ai announced a "Longevity Intelligence Infrastructure" that unifies diverse biological and clinical datasets to decipher the "language of life" and predict interventions [45] [46]. By integrating genomic, epigenetic, clinical, and behavioral data, such platforms aim to identify hidden aging biomarkers and test interventions virtually (via **digital twin** simulations) to see how they might extend healthspan [47] [48]. In sum, AI is rapidly becoming an indispensable tool in longevity science, used to discover drug candidates, build more accurate aging clocks, and personalize anti-aging strategies.

- **Biomarkers and Aging Clocks:** Another key tool in longevity research is the development of better **biomarkers** – measurable indicators of biological aging and functional capacity. This week's discussions reinforced that aging biomarkers are moving from theory to practice. Scientists are refining **epigenetic clocks** (which measure DNA methylation patterns) to more accurately gauge an individual's "biological age" and predict their risk of functional decline [49]. For example, the European DO-HEALTH trial (cited in a recent analysis) showed that a combination of vitamin D, omega-3, and exercise not only reduced frailty in older adults but also **slowed epigenetic aging**, demonstrating that healthspan can be quantified and improved in a measurable way [50]. Beyond DNA, large proteomic studies (e.g. UK Biobank data) are yielding **protein signatures** that correlate with aging and mortality risk [49]. Digital health is contributing too: wearables and smartphones can capture gait speed, activity levels, heart rate variability, etc., providing real-time "digital biomarkers" of resilience or decline [49]. This past week, experts argued that such **healthspan metrics** – frailty indexes, cognitive function tests, biomarker panels – should be tracked alongside traditional survival in trials [51] [52]. The ultimate goal is to have reliable, multi-factorial biomarkers that can rapidly tell if an intervention is truly keeping someone biologically younger or functionally fitter, without waiting decades. Recent advances in **AI are aiding biomarker development** too, by processing complex patterns (for instance, AI can integrate epigenetic data with lab tests and imaging to yield a composite aging score). Having these tools will greatly accelerate geroscience research and drug development, as scientists can sooner see whether a therapy is hitting its mark in extending healthspan.

- **Advanced Imaging and Screening Platforms:** Longevity research also benefits from cutting-edge lab techniques. High-resolution imaging and single-cell sequencing are illuminating aging at the cellular level. In the glial cell study, researchers used **fluorescent microscopy** to watch how knocking out a single gene in worm glia ramped up autophagy throughout the organism and prevented toxic protein build-up in neurons [53]. They coupled this with **RNA sequencing** to map how gene expression shifted to an anti-aging profile [54]. Similarly, other studies are using technologies like **senescence-associated beta-gal staining** and mass cytometry to identify senescent cells in tissues and quantify how well senolytic drugs clear them. On the clinical side, improved imaging modalities (e.g. MRI techniques to gauge muscle quality or brain amyloid in aging) act as early readouts for functional decline or improvement. This week also highlighted the importance of **platform technologies** in accelerating trials – for instance, specialized assays to measure glycocalyx integrity in blood vessels helped validate the effect of the HMW-HA supplement on mouse muscle function [18] [19]. Even in drug development, new screening systems like **organoids** (mini-organs grown from stem cells) are being used to test longevity interventions on human-like tissues. Overall, the convergence of AI, sophisticated biomarkers, and high-throughput biology is giving researchers unprecedented power to probe aging and test interventions quickly and reliably.

Ethical and Practical Considerations

As longevity science races ahead, it brings to the fore several ethical and practical issues that were echoed in this week's discussions:

- **Safety vs. Benefit:** A foremost consideration is ensuring that interventions designed to slow aging **do not introduce new risks**. Many of the approaches are double-edged: for instance, manipulating the TOR pathway can extend lifespan in animals, but over-inhibiting TOR could impair wound healing or immune function. The Rapalink-1 findings caution that even seemingly beneficial supplements like agmatine could be harmful in the wrong context – the researchers noted that agmatine's effects depend on metabolic conditions and that indiscriminate use might **"contribute to certain pathologies"** [41]. Similarly, the senolytic strategy (killing senescent cells) must be handled carefully; the Rubedo trial news underscored that targeting senescent cells via ferroptosis is promising but could harm normal cells if not specific enough [55]. Any new "anti-aging" drug will need rigorous testing for side effects across various organ systems, especially since they may be taken by relatively healthy older individuals for prevention. In short, *first do no harm* remains paramount – extending life or healthspan is pointless if the intervention causes unacceptable toxicity.

- **Ethical use of advanced therapies:** The prospect of restoring function in coma patients or rejuvenating the aged brain raises hope, but also difficult ethical questions. For example, if senolytic/stem cell therapy can partly reverse coma, when should it be applied, and who decides on treating a patient who cannot consent? The line between living and end-of-life could blur if technology offers a chance of "reawakening" long-term unconscious patients, so society will need to debate guidelines. Additionally, many longevity interventions (like gene therapies, bespoke cell infusions) are high-tech and initially expensive. This raises concerns about **equitable access** – will these advances be available only to the wealthy, potentially exacerbating health disparities? Contributors this week emphasized that as we pursue healthy aging, we must ensure **accessibility** and fairness, lest we create a world where only some can afford extra healthy years [56]. Regulatory bodies and healthcare systems will face pressure to approve and cover anti-aging treatments, and they'll have to

weigh cost-effectiveness (though one analysis noted every $1 invested in healthy aging returns $3 in economic benefit [57]).

- **Regulation and Oversight:** Longevity science is entering a phase where traditional regulatory frameworks are being tested. The FDA approval of Forzinity under the **accelerated approval** pathway shows regulators' willingness to endorse therapies for unmet needs, but it also means requiring follow-up trials and vigilant monitoring [58]. As more companies seek approval for aging-targeted drugs (which may target multiple conditions at once), the FDA and other agencies might need new criteria to evaluate **"geroprotectors."** There's ongoing debate about recognizing aging itself as a treatable condition – the outcome of trials like TAME (metformin in aging) could influence policy. Ethically, we must avoid hype and ensure that interventions are sold based on solid evidence of benefit. The scientific community is calling for robust clinical trials and transparent reporting for longevity interventions, to counteract the many unproven anti-aging products already on the market. **Data privacy** is another practical concern: as AI-driven platforms like Elivion integrate personal health data, safeguarding individuals' genetic and health information is critical [45] [59]. Striking the right balance between innovation and protection (of people and their data) will be key as this field progresses.

- **Societal Impact:** Finally, there are broad societal questions. If we succeed in significantly extending healthy lifespan, how do we ensure quality of life in those extra years and the sustainability of healthcare systems? The focus on healthspan (not just lifespan) addresses part of this – it's about preventing disability, so older individuals remain independent and engaged, potentially reducing long-term care burdens. However, greater numbers of healthy, longer-living people will affect retirement ages, workforce dynamics, and intergenerational responsibilities. Ethicists highlight the importance of **inclusive dialogue** as longevity therapies develop: different cultures may have varying attitudes toward prolonging life, and we must consider the psychological and social implications of pushing the boundaries of human lifespan. Encouragingly, the discourse is already changing – this week's Milken Institute update noted that governments from Japan to Singapore are incorporating **healthy longevity into policy**, aiming to keep populations productive and vibrant longer [60]. The pursuit of "immortality" or extra decades of life is not just a medical quest but a social one, requiring us to confront our views on aging, equity, and what it means to live well.

Future Directions

The flurry of findings this week illuminates an exciting road ahead in longevity science. Here are some anticipated **next steps and trends** emerging from the current discoveries:

- **Translating lab results to human therapies:** A clear future direction is moving promising interventions from animal models into human trials. For example, after Rapalink-1's success in yeast, the researchers plan to test its effects on human cells – including cells from cancer or other patients where TOR is overactive – to see if modulating agmatine metabolism has similar benefits [61]. If results are positive, we might eventually see Rapalink-1 or related compounds enter clinical trials for age-related diseases (or even general age-delaying purposes). Similarly, the glial cell alkalinization concept may prompt studies in mammals. We could envision experiments to raise glial pH in mouse models of aging or Alzheimer's to assess lifespan and cognitive outcomes. The glycocalyx study in mice is primed for translation: since HMW-HA is available as a supplement, a logical next step is a **clinical trial in older adults** to see if it can improve fitness or reduce frailty (with careful monitoring

of vascular markers) [62]. Such trials would measure whether preserving the glycocalyx in humans yields better muscle function or aerobic capacity in aging, directly testing the approach in our own species.

- **Advancing clinical trials for healthspan extension:** On the clinical front, expect to see more trials that explicitly measure **healthspan outcomes** (not just disease endpoints). The FDA's acceptance of muscle function as a surrogate in the Forzinity approval sets a precedent – future anti-aging trials might be approved based on improvements in physical performance, cognitive function, or biological age markers, rather than having to prevent a specific disease. One imminent example is the TAME (Targeting Aging with Metformin) trial, which is testing whether an older drug can delay the onset of multiple age-related diseases. Its results, when available, could influence regulatory thinking on **"aging as an indication."** Likewise, the field is eagerly awaiting results from trials of senolytics in humans (e.g. UNITY's UBX1325 in diabetic macular edema, or other studies in osteoarthritis) to see if clearing senescent cells translates to functional benefits like improved vision or less pain. The next few years will likely bring **combination trials** too – since aging is multi-factorial, combining interventions (for instance, a senolytic drug plus a mitochondrial booster, or diet plus a metformin-like pill) might have additive effects. Indeed, an analysis this week noted that stacking simple interventions (vitamin D, omega-3, exercise) produced measurable aging slowdown [50]; future regimens may layer drugs, supplements, and lifestyle into a comprehensive longevity protocol.

- **Integrating technology and personalized medicine:** As longevity therapies progress, **personalization** will become a focus. Given tools like epigenetic clocks and AI predictors of aging, doctors might tailor interventions to an individual's aging profile. We can foresee a scenario where a person's data (genomic, metabolic, imaging, wearable) is fed into an AI platform which then suggests the optimal combination of therapies to maximize their healthspan. This could include recommending existing medications (repurposed for aging), specific dietary plans, or enrolling the person in a particular rejuvenation therapy trial. The multi-agent AI systems demonstrated (like K-Dense) will continue to improve, possibly giving researchers instantaneous feedback on which biomarkers to track and which patients benefit most from an intervention. On the biotech side, more companies will use AI to discover **new geroprotective compounds** and identify novel aging pathways (the success of approaches like ALEMBIC and Elivion's platform will spur competitors). We may also see **biomarker-driven endpoints** in trials – for instance, therapies might be approved if they significantly slow epigenetic aging or reduce inflammatory age markers, alongside clinical outcomes.

- **Holistic approaches and functional rejuvenation:** The ultimate goal of longevity research is not just to add years to life, but to achieve some level of **biological rejuvenation** – restoring the function of tissues or systems that have aged. Some news this week hints at that paradigm: the coma reversal concept is essentially trying to regenerate a severely damaged aging brain to a more "youthful," functional state. In the future, more regenerative therapies (like plasma-derived factors, stem cell infusions, or gene editing) will likely be tested in the context of aging. We anticipate progress in things like **partial cellular reprogramming** (using Yamanaka factors or similar to reset epigenetic age in cells) – currently in preclinical stages at labs and companies such as Altos – potentially moving into animal models and then human trials for specific age-related conditions. There is also interest in **organ-specific rejuvenation**: for example, therapies to rejuvenate the immune system (like thymus regeneration or clearing aged immune cells) or the brain (perhaps via

young CSF infusions, as some Stanford studies have explored). Over the next few years, each hallmark of aging (from senescence to telomere shortening to mitochondrial dysfunction) could have at least one therapy in human testing.

- **Anticipated impact on public health:** If even a few of these emerging interventions prove successful, the impact on society could be profound. Enhancing healthspan means more people in their 70s, 80s, and beyond who are **active and independent**, which would transform healthcare (less burden of chronic disease) and economies (a more able older workforce). Experts are already forecasting that in the next five years, metrics of healthspan will start appearing alongside lifespan in policy and investment decisions [63]. In the best-case scenario, we might achieve what gerontologists call "compression of morbidity" – concentrating illness and decline into a shorter period at the end of life, with the majority of added years being vigorous ones. The discoveries from this week – if they continue to develop – each contribute a piece to that puzzle: a drug to keep metabolism youthfully tuned, a way to preserve muscle and mobility, a strategy to rejuvenate the brain or immune system, and technology to detect problems early and guide interventions.

In summary, the past week's breakthroughs underscore that **longevity science is maturing** rapidly. Early research is unraveling new targets (lysosomal signaling across generations, glial ion channels, metabolic feedback loops), while translational efforts are bringing first-of-their-kind therapies to patients (mitochondrial drugs, senolytic trials). We are likely on the cusp of an era where extending healthspan becomes an achievable medical objective. The coming years will determine how these innovations coalesce into strategies that not only help us live longer, but allow us to truly **thrive longer**, preserving the vitality and consciousness that make life worth living. Each step – scientific, clinical, ethical – will shape a future in which aging could become a more manageable, even reversible, aspect of the human condition [64] [39].

Sources:

- Zhang et al., *Science*, Oct 2025 – Lysosomal signals epigenetically inherited across generations [65] [66].
- Borfitz, D. "TOR Inhibitors Could One Day Be Multi-Purpose Healthspan Drugs." *Bio-IT World*, Oct 14, 2025 [67] [5].
- Kumar et al., *Communications Biology*, 2025 – Rapalink-1 extends yeast lifespan via agmatinase metabolic feedback [68] [2]; ScienceAlert coverage by D. Nield, Oct 9, 2025 [1] [4].
- Hanson, C. "Worms and Aging: How Non-Neural Cells... Spur Longevity." *InventUM (U. Miami)*, Oct 10, 2025 [8] [12].
- Bianchi et al., *Science Advances*, 2025 – Glial alkalinization extends C. elegans lifespan and healthspan [9] [12].
- Impact Journals Press Release (EurekAlert), Oct 14, 2025 – Glycocalyx-targeted therapy prevents age-related muscle loss [16] [17].
- News-Medical. "Targeting the glycocalyx may reduce frailty in older adults," Oct 14, 2025 [15] [69].
- Immorta Bio Press Release (PR Newswire), Oct 14, 2025 – Senolytic + stem cell approach for coma [23] [24].
- Bioengineer.org. "Reversing Coma: Senolytics and Stem Cells Lead Future," Oct 15, 2025 [25] [28].
- Reuters. "US FDA approves first treatment for rare disease in males (Barth syndrome)", Sept 19, 2025 [33] [58].
- Longevity.Technology. "Is this mitochondria's big moment?", Oct 9, 2025 [31] [39].
- Bystritskaya, A. "From biomarkers to balance sheets," *Longevity.Technology*, Oct 14, 2025 [49] [50].

- Garth, E. “Rubedo gains FDA clearance for senotherapeutic trial,” *Longevity.Technology*, Sept 17, 2025 [44] [55].
- Sullivan, D. “New AI tool… accelerate longevity research,” *Longevity.Technology*, Sept 18, 2025 [42] [43].
- Elite Labs (via Tokenist). “Elivion AI… New Era of Longevity AI,” Oct 10, 2025 [45] [47].
- ScienceAlert. “A New Class of Drug… Fights Aging on a Cellular Level,” Oct 9, 2025 [7] [41].
- Bioengineer.org. “Reversing Coma” – ethical and societal commentary [56] [30].

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[1] [3] [4] [6] [7] [41] A New Class of Drug Created That Fights Aging on a Cellular Level : ScienceAlert
https://www.sciencealert.com/a-new-class-of-drug-created-that-fights-aging-on-a-cellular-level

[2] [68] New drug and enzyme class found to have anti-ageing properties | EurekAlert!
https://www.eurekalert.org/news-releases/1099727

[5] [61] [67] TOR Inhibitors Could One Day Be Multi-Purpose Healthspan Drugs
https://www.bio-itworld.com/news/2025/10/14/tor-inhibitors-could-one-day-be-multi-purpose-healthspan-drugs

[8] [9] [10] [11] [12] [13] [14] [53] [54] Worms and Aging: How Non-Neural Cells in the Central Nervous System Could Spur Longevity - InventUM
https://news.med.miami.edu/worms-and-aging-how-non-neural-cells-in-the-central-nervous-system-could-spur-longevity/

[15] [20] [21] [62] [69] Targeting the glycocalyx may offer a new approach to reduce frailty in older adults
https://www.news-medical.net/news/20251014/Targeting-the-glycocalyx-may-offer-a-new-approach-to-reduce-frailty-in-older-adults.aspx

[16] [17] [18] [19] [22] [40] Supplement that supports the glycocalyx in blood vessels may reduce frailty in older adults | EurekAlert!
https://www.eurekalert.org/news-releases/1101696

[23] [24] [27] [29] Immorta Bio Publishes in Prestigious Academic Journal Patent-Pending Approach to "Re-Awakening" Coma Patients
https://www.prnewswire.com/news-releases/immorta-bio-publishes-in-prestigious-academic-journal-patent-pending-approach-to-re-awakening-coma-patients-302583424.html

[25] [26] [28] [30] [56] [64] Reversing Coma: Senolytics and Stem Cells Lead Future
https://bioengineer.org/reversing-coma-senolytics-and-stem-cells-lead-future/

[31] [35] [36] [37] [38] [39] Is this mitochondria’s big moment?
https://longevity.technology/news/is-this-mitochondrias-big-moment/

[32] [33] [34] [58] US FDA approves first treatment for rare, life-threatening disease in males | Reuters
https://www.reuters.com/business/healthcare-pharmaceuticals/us-fda-approves-first-treatment-rare-life-threatening-disease-males-2025-09-19/

[42] [43] New AI tool demonstrates potential to accelerate longevity research
https://longevity.technology/news/new-ai-tool-demonstrates-potential-to-accelerate-longevity-research/

[44] [55] Rubedo gains FDA clearance for senotherapeutic trial
https://longevity.technology/news/rubedo-gains-fda-clearance-for-senotherapeutic-trial/

[45] [46] [47] [48] [59] Elivion AI Unlocks the ‘Language of Life,’ Ushering in a New Era of Longevity AI
https://markets.financialcontent.com/buffnews/article/tokenring-2025-10-10-elivion-ai-unlocks-the-language-of-life-ushering-in-a-new-era-of-longevity-ai

49 50 51 52 57 60 63 From biomarkers to balance sheets
https://longevity.technology/news/from-biomarkers-to-balance-sheets/

65 66 Leaving a mark: New research shows how longevity is inherited across generations
https://phys.org/news/2025-09-longevity-inherited-generations.html