The Arising

HEALTH INTELLIGENCE / JOHN GRIFFIN, MD

NOVEMBER 11, 2026 · EDITED READER EDITION

Strength, Heart Health & Emerging Science

40 reviewed articles on health, longevity and the discoveries changing what comes next.



This edited reader edition brings together 40 reviewed articles from the November 11 source. Seven of the original 47 stories are not included pending further review. It is not a reproduction of the complete original issue. Research briefs are excluded.

The articles retain their source dates, evidence limits and links. This edition was assembled November 12, 2026.

Read the living collection

Inside this edition

01 Weight Training Reduced Lean Tissue Loss on Tirzepatide

02 FDA Orders Vision-Loss Warning on Every GLP-1 Drug Label

03 Review Suggests Eating Less Protein May Slow Aging

04 Five-Year Data: Earlier Vutrisiran Linked to Fewer Deaths and Heart Events

05 Earlier Bedtime Linked to More Weight Loss in Prediabetes Sleep Trial

06 Probiotics Linked to Better Cognitive Test Scores in Mild Impairment and Alzheimer's, Meta-Analysis Finds

07 Hyperbaric Oxygen Linked to VO2 Max Gains, but Study Lacked Controls

08 Model Links Harsher Breast Cancer Chemotherapy to Earlier Aging-Related Disease

09 Triglyceride-Rich Particles and Lp(a) Share Blood Protein Signals of Artery Inflammation

10 Vesicles Shed by Mitochondria Carry Microglial Protein Ggt1 Into Alzheimer's Pathology

11 Implanted Vagus Nerve Stimulator Linked to Sustained Responses in Rheumatoid Arthritis at Two Years

12 Beam's Base-Edited Sickle Cell Therapy Meets Main Goal, FDA Filing Planned

13 Home Exercise Improved Cancer Survivors' Peak Oxygen Uptake, Heart Protection Unproven

14 Company Reports Zabopegdutide Resolved MASH in 63% in Phase 2 Biopsy Data

15 Late Chronotype Linked to Metabolic Risk and Gut-Barrier Differences

16 Urolithin A Supplement Failed to Improve Artery Function in Adults With Obesity

17 Faster Everyday Walking Pace Linked to Lower Death Risk in Older Adults

18 UK Launches £20 Million AI Hub for Earlier Dementia Diagnosis

19 Review Links Gut Bacteria to Parkinson's Inflammation and Levodopa Availability

20 Sironax to Show Preclinical Data on NAD-Pathway Obesity Pill

21 Alpha-1 Liver Disease Gets a Shared Definition as RNA Editing Data Emerge

22 Oura Agrees to Acquire AI Care Startup Counsel Health

23 Humacyte Begins First Human Trial of Lab-Grown Coronary Bypass Vessel

24 Tai Chi and Exercise Classes Barely Moved Sleep in Older Cancer Survivors

25 Short-Lived mRNA Therapy Helped Mouse Hearts Repair After Heart Attack

26 Accurate AI Health Answers May Still Steer People Toward Worse Choices

27 Elysium Health Plans Six Additional Peptides Under New HHS Policy

28 OIG Finds Indiana Public Entities Recouped $205 Million Intended for Nursing Homes

29 Ionis Cuts Tryngolza Sales Forecast by Roughly Half

30 Long-Term Unemployment Reaches 32% of Jobseekers Aged 55 and Older, AARP Data Show

31 Fat Molecules Get Longer With Age Across Species, and Reversing It Extended Worm Lifespan

32 Two Brain-Aging Studies Are Listed: Baltimore PET Imaging and a VR Driving Game

33 Evipedia Puts 860-Plus AI-Written Longevity Evidence Reviews on Hugging Face, Free to Download

34 Deno's Team Joins Cloudflare, and Profitable Oxide Raises $445 Million

35 Big Pharma Filled the Speaker List at a Major Aging-Drug Meeting

36 Google Launches Gemini 4 Argon; AI Weather Models Add a Day of Warning

37 Frontier AI Prices Fell Sharply in Weeks, Changing What Medical AI Can Afford

38 Uranium Slowly Loses Its Easy Mode, and the Physics Says the Clock Is Real

39 Pioneer Labs Reports Bioplastic Production From Simulated Mars Resources

40 Did Storable Grain Make Conquered Land More Valuable to Early States? A Clear Claim With a Clear Test

01 / LONGEVITY & HEALTHSPAN

Weight Training Reduced Lean Tissue Loss on Tirzepatide

Losing more than 20 kilograms on tirzepatide also means losing lean tissue, and a new trial shows that lifting weights three times a week reduced that loss and turned a fall in strength into a gain.



Photo: “Marines' voices echo, prompt new gym hours 150927-M-AI083-067” by Lance Cpl. Jason Jimenez, Public domain

The study, published in Nature Medicine, was a randomised controlled trial of 120 adults with overweight or obesity. All were treated with tirzepatide, a weight-loss drug sold as Mounjaro. Half were randomly assigned to resistance exercise three times a week for 40 weeks. The other half carried on with usual activities. Adherence to the exercise was 91%, and most participants tolerated increases in the drug dose up to 15 mg weekly.

Weight loss was nearly identical. The drug-only group lost 22.61 kg and the exercise group lost 22.28 kg. Fat loss was also similar, at 17.19 kg and 17.98 kg.

The difference showed up in lean soft tissue, which includes muscle, organs, skin and body water but excludes bone mineral. The drug-only group lost 4.67 kg of it. The exercise group lost 3.57 kg, a reported between-group difference of 1.09 kg (95% confidence interval 0.43 to 1.75 kg). That is about 24% less lean tissue lost, calculated from the rounded group averages. Loss of lean mass in the arms and legs was also smaller with exercise: 2.92 kg against 3.55 kg.

Strength moved in opposite directions. With tirzepatide alone, bench press fell by 4.09 kg, lat pulldown by 4.42 kg and knee extension by 4.42 kg. With exercise added, they rose by 4.68 kg, 8.58 kg and 5.88 kg. The university's coverage puts the change at about 10% up for the trained group and 10% down for the others.

Participants were aged 50 to 70, according to the university's coverage, and did not have type 2 diabetes. Lead investigator Danny Green said loss of lean mass "is an inevitable part of weight loss by any method". "We now know that muscle loss can be reduced and strength increased when regular resistance training is used alongside tirzepatide treatment," he said. He argues resistance exercise should be part of the prescription for people on weight-loss drugs.

Green also said the goal should be better health and function, not just a lower number on the scales. In theory, he said, that should translate to a longer healthspan, meaning more years lived independent and free of chronic disease. The trial itself measured body composition and strength, not years of healthy life.

The university says the drug's manufacturer supplied tirzepatide free but had no part in the design, running or evaluation of the trial.

Next, watch for longer follow-up that shows whether the muscle and strength preserved during weight loss translate into healthier years, as Green expects.

Sources

Source 1: https://pubmed.ncbi.nlm.nih.gov/43003902/

Source 2: https://doi.org/10.1038/s41591-026-04752-5

Read this article online

02 / LONGEVITY & HEALTHSPAN

FDA Orders Vision-Loss Warning on Every GLP-1 Drug Label

The FDA is requiring every maker of a GLP-1 receptor agonist to add a warning about a rare eye condition that can cause sudden, painless vision loss in one eye.

The condition is called nonarteritic anterior ischemic optic neuropathy, or NAION. It involves reduced blood flow to the optic nerve, which carries signals from the eye to the brain. According to the FDA's drug safety communication, cases have been reported for all currently marketed GLP-1 therapies.

The agency began its review after a 2024 study in JAMA Ophthalmology, led by Jimena Tatiana Hathaway, found a potential link between semaglutide and NAION.

The FDA then pooled data from 126 clinical trials of GLP-1 drugs. Among 78,217 patients who received a GLP-1 therapy, there were 10 probable cases of NAION. Among 56,036 patients who received a comparator, there were five probable cases, all in people on placebo. None appeared in those taking a non-GLP-1 glucose-lowering drug.

That works out to 6.4 events per 100,000 person-years with GLP-1 therapy, against 3.8 per 100,000 person-years with a comparator, according to the communication.

The FDA also ran a retrospective cohort study using its Sentinel data network. It followed people with type 2 diabetes who started a GLP-1 drug, an SGLT2 inhibitor or a DPP-4 inhibitor between 2019 and 2024. GLP-1 users had a higher risk of NAION than those on a DPP-4 inhibitor. The difference against SGLT2 inhibitor users was not statistically significant.

For people taking these drugs, the FDA's guidance centers on speed. Health care professionals should tell patients to report immediately any sudden, painless drop in vision, loss of part of the visual field, or visual disturbance in one eye. If NAION is suspected, the FDA wrote, "promptly refer the patient to an ophthalmologist for evaluation." It added: "Consider discontinuing the GLP-1 receptor agonist in patients who develop NAION, unless the benefits of treatment clearly outweigh the risk."

The FDA describes a potential increased risk, not a proven causal mechanism. Patients who experience sudden visual symptoms should contact their health care professional right away and discuss treatment changes with them.

Sources

Source 1: https://www.fda.gov/drugs/drug-safety-communications/fda-requires-safety-labeling-changes-glucagon-peptide-1-receptor-agonists-glp-1-ras-due-potential

Read this article online

03 / LONGEVITY & HEALTHSPAN

Review Suggests Eating Less Protein May Slow Aging

Protein is being added to cereal, coffee and bottled water, and older adults are routinely urged to eat more of it, yet a new review argues that the evidence for "more is better" is far less settled than the shelf space suggests.



Photo: by Shixart1985, CC BY 2.0

The paper, published in Cell Press Blue in 2026, is a review written by Knopf and Lamming. It draws on work in yeast, flies, rodents and people. Its central argument is that lower-protein diets promote metabolic health, healthspan and lifespan across diverse organisms.

The starting point is a gap between advice and data. The official Recommended Dietary Allowance for protein is 0.8 grams per kilogram of body weight, for nonpregnant adults. Intakes of 1.0–1.2 grams per kilogram are routinely recommended for people over 65 to prevent sarcopenia, the age-related loss of muscle, and frailty. The review reports that the most recent Dietary Guidelines for America suggest 1.2–1.6 grams per kilogram. According to a 2025 report cited in the review, 61 percent of American consumers increased their protein intake in 2024, up from 48 percent in 2019.

The authors set against this a body of evidence pointing the other way. In rodents, low-protein, high-carbohydrate diets extend lifespan and improve metabolic health. Protein restriction also increases the lifespan of yeast and flies. In rodents, it raises energy expenditure and cuts fat even though the animals eat more, and it still works when started late in life, at 18 to 20 months of age in mice.

Human data are mixed. Observational studies cited in the review link high-protein diets to a greater risk of diabetes, cancer and death, including death from cardiovascular events. An analysis of US national survey data found higher protein consumption tracked with higher mortality and more age-related disease. A 2023 UK study found that higher protein intake was positively associated with sarcopenia in older twins, which cuts against the usual advice.

Randomised trials point to metabolic effects of eating less protein. In a 2016 trial from the authors' group, people on a low-protein diet for 43 days had lower body weight, less fat mass and lower fasting blood glucose despite eating more calories. A five-week intervention in lean men improved insulin sensitivity and raised energy expenditure. In patients with metabolic syndrome, 27 days of protein restriction reduced fat, improved insulin sensitivity and lowered glucose, lipids and inflammation. Conversely, a high-protein, low-carbohydrate diet was less effective than a moderate-protein diet at lowering blood glucose and promoting prediabetes remission.

The review points to a hormone, fibroblast growth factor 21 (FGF21), as a central player. Protein restriction robustly raises FGF21 in mice, rats and humans. In mice, the hormone drives much of the response: animals engineered to make extra FGF21 lived about 30 percent longer in males and about 40 percent longer in females. Mice lacking the gene did not live longer on a low-protein diet. These are mouse findings; the lifespan question has not been answered in people.

The authors also describe two cellular nutrient-sensing pathways, GCN2 and mTOR. Low amino acid levels activate GCN2 and inhibit mTORC1, a master sensor of nutrients. In mouse knockout models, the benefits of protein restriction were lost without that mTORC1 inhibition.

Not all protein is equal in this account. Restricting just three amino acids, methionine, isoleucine and valine, recapitulates many of the benefits of protein restriction, according to the paper. That makes the amino acid makeup of the diet, not only the total amount, a key factor in aging.

On who should care, the evidence is not uniform. The paper acknowledges the case for higher protein in older adults, especially paired with resistance exercise, to combat muscle loss and frailty. In coverage by ScienceDaily, corresponding author Dudley Lamming said: "It's absolutely crystal clear that there are benefits of protein to muscle growth and exercise response of active individuals. But because most people are relatively sedentary, many people are likely consuming more protein than they actually need, which probably has negative health consequences."

Next to watch are longer human trials that measure muscle function alongside metabolic health. This review does not establish that eating less protein slows aging or extends life in people.

Sources

Source 1: https://pubmed.ncbi.nlm.nih.gov/42639474/

Source 2: https://pmc.ncbi.nlm.nih.gov/articles/PMC13501984/

Source 3: https://doi.org/10.1016/j.cpblue.2026.100079

Read this article online

04 / LONGEVITY & HEALTHSPAN

Five-Year Data: Earlier Vutrisiran Linked to Fewer Deaths and Heart Events

Patients with a heart condition who stayed on one drug for five years had roughly 41% to 48% lower risk of death or repeat cardiovascular events than patients who started it later, according to new trial data.

Alnylam presented the five-year results from its HELIOS-B trial at the American Heart Association Scientific Sessions 2026, according to the company announcement. The drug is AMVUTTRA (vutrisiran). It is approved by the FDA for transthyretin amyloid cardiomyopathy, known as ATTR-CM, and for hATTR polyneuropathy. It is given every three months.

The analysis included a two-year open-label extension. It compared patients treated continuously with those who began treatment later. Outcomes were tracked through 60 months.

In the monotherapy group (395 patients), continuous treatment was linked to a 48% lower risk of the combined outcome of all-cause death and recurrent cardiovascular events (hazard ratio 0.515; 95% confidence interval 0.368 to 0.722). Recurrent cardiovascular events alone were 50% lower (rate ratio 0.501).

In the overall population (654 patients), the reductions were 41% for the combined outcome (hazard ratio 0.589) and 41% for recurrent events (rate ratio 0.587).

The company also reported gains in Kansas City Cardiomyopathy Questionnaire scores, a measure of how patients feel and function with heart disease. Two blood markers of heart strain and injury, NT-proBNP and troponin I, stayed stable. Safety through 60 months was consistent with prior experience, according to the company.

A two-year, single-center post hoc exercise analysis of 48 patients showed preserved peak oxygen uptake, with a difference of 1.57 mL/kg/min versus placebo (95% confidence interval 0.14 to 3.00; p=0.031). Peak oxygen uptake reflects how much oxygen the body can use during hard exercise.

The company states that the reported P values are nominal and were not adjusted for multiple comparisons.

These are company-reported figures from an extension in which patients were compared by when they started treatment, not a new randomized comparison.

For readers following ATTR-CM, the relevance is timing: patients treated continuously fared better through 60 months than those who began treatment later.

Next to watch is the full presentation and any peer-reviewed publication of the HELIOS-B five-year analyses.

Sources

Source 1: https://investors.alnylam.com/press-release?id=65c2d0ab-27cd-42a0-a174-728416129efe

Read this article online

05 / LONGEVITY & HEALTHSPAN

Earlier Bedtime Linked to More Weight Loss in Prediabetes Sleep Trial

Adding sleep coaching did not significantly improve blood sugar or weight loss compared with lifestyle coaching alone. An exploratory analysis linked earlier sleep onset with greater weight loss.

The finding comes from a randomized controlled trial of 196 adults with A1c-confirmed prediabetes and short sleep confirmed by wrist-worn activity monitors, published in the journal Nursing Research in 2026. Susan Kohl Malone and colleagues compared a lifestyle program with the same program plus a sleep intervention.

Short sleep is a modifiable barrier to diabetes prevention, the authors write. Yet no adequately powered trial had tested whether building a behavioral sleep intervention into a lifestyle program improves blood sugar or weight loss in this group.

Both programs lasted eight weeks and were delivered one-on-one by video call. Before and after, participants spent two weeks at home wearing a continuous glucose monitor and an activity monitor that estimates sleep. The main outcome was the share of time sensor glucose sat above 140 mg/dL.

On that outcome, the two groups did not differ significantly. Both groups slept more and lost weight by the end, with no significant difference between them on either measure.

The sleep group did report gains the lifestyle-only group did not match. Their self-reported sleep quality and daytime sleepiness improved significantly more. Their sleep onset also moved earlier, a change that was significant only in the sleep group, although the difference between groups did not reach statistical significance.

The weight result came from a post hoc analysis, meaning it was run after the main results, across all participants regardless of group. Earlier sleep onset was independently associated with greater weight loss. Changes in sleep duration were not.

According to Medical Xpress, which covered the study for NYU, each hour earlier that people fell asleep was associated with about 1.3 pounds (0.6 kilograms) of weight loss over the eight weeks.

"When we fall asleep—not just how much we sleep—seems to be an important and overlooked factor for weight loss and metabolic health," Malone said, as quoted by Medical Xpress. She also told the outlet that falling asleep earlier may limit late-night eating and better align food intake with the body's optimal metabolic window.

The authors write that circadian timing, the body's daily rhythm, is a promising and modifiable target for diabetes prevention. They call for prospective trials designed to test sleep timing interventions on metabolic outcomes, and those trials are the next thing to watch.

Sources

Source 1: https://pubmed.ncbi.nlm.nih.gov/42910621/

Source 2: https://doi.org/10.1097/NNR.0000000000000957

Source 3: https://nursing.nyu.edu/w/news/press-release/earlier-bedtime-linked-weight-loss-prediabetes

Read this article online

06 / LONGEVITY & HEALTHSPAN

Probiotics Linked to Better Cognitive Test Scores in Mild Impairment and Alzheimer's, Meta-Analysis Finds

Across ten randomized trials, people with mild cognitive impairment or Alzheimer's disease who took probiotics or synbiotics scored higher on thinking tests than those given placebo or usual care. The authors rated the evidence very low certainty.

The finding comes from a systematic review and meta-analysis published in Ageing Research Reviews in 2026 by Zhang M, Zhang H, Fu B and Xu D. A meta-analysis pools results from several earlier studies into one estimate. This one combined ten randomized controlled trials with 739 participants in total.

The authors searched four databases: PubMed, Embase, Web of Science and the Cochrane Library. They included trials that compared live probiotics or synbiotics with placebo or usual care. A synbiotic pairs a probiotic with a prebiotic, a food source for gut microbes. The participants had mild cognitive impairment, Alzheimer's disease or related cognitive decline.

Because trials use different cognitive tests, the team expressed each result as a standardized mean difference. This puts all the scores on a common scale. The pooled difference was 0.50, with a 95% confidence interval of 0.19 to 0.81, in favor of probiotics or synbiotics. The authors describe this as a possible improvement in cognitive function.

The trials did not agree closely. The authors report substantial variation between them (I^2 of 76%). The authors rate the certainty of the evidence as very low, and the range expected for a future setting, from -0.57 to 1.57, includes no benefit at all.

For anyone wondering which product might matter, the analysis offers no clear answer. The authors found no statistically significant differences in effect by disease category, length of treatment, type of intervention, average age of participants, strain mix, dose or the cognitive test used. Sensitivity analyses kept the direction and statistical significance of the pooled effect. A test for small-study effects, which can distort pooled results, did not detect significant small-study effects (P = 0.318).

The authors conclude that probiotics or synbiotics may improve cognitive test performance. They also call for larger, well-designed randomized trials.

Next to watch is whether larger trials with consistent methods show that the test-score gain holds up, and for whom.

Sources

Source 1: https://pubmed.ncbi.nlm.nih.gov/43003143/

Source 2: https://doi.org/10.1016/j.arr.2026.103442

Read this article online

07 / LONGEVITY & HEALTHSPAN

Hyperbaric Oxygen Linked to VO2 Max Gains, but Study Lacked Controls

Among 1,032 patients who completed a course of hyperbaric oxygen therapy, average peak aerobic capacity, the best-known measure of fitness in ageing research, was higher after treatment than before.



Photo: “40" Mild Hyperbaric Chamberexternal” by Mckeeman at English Wikipedia, CC BY 3.0

The finding comes from a retrospective cohort study in Frontiers in Aging, drawn from consecutive patients treated between 2018 and 2025. The authors are affiliated with the Sagol Center for Hyperbaric Medicine and Research at Shamir Medical Center in Israel. All patients had a maximal or peak-effort exercise test before and after therapy.

Hyperbaric oxygen therapy, or HBOT, means breathing 100% oxygen at pressures above normal air pressure. The protocol studied used 2.0 atmospheres absolute with intermittent air breaks, over a mean of 58.8 sessions. The authors describe it as based on the hyperoxic–hypoxic paradox. They say earlier controlled trials of the same protocol reported fitness gains in healthy athletes and older adults.

Peak aerobic capacity, called VO2 max, rose by an average of 2.29 mL per kilogram per minute (95% confidence interval 2.10 to 2.47). Oxygen uptake at the first ventilatory threshold, a submaximal marker linked to the effort sustainable in daily activity, rose by 2.12. All seven prognostic exercise-test measures improved (all $p < 0.001$).

Oxygen pulse, which reflects how much oxygen each heartbeat delivers, rose by 0.79 mL per beat. The VE/VCO2 slope, a measure of breathing efficiency in which lower is better, fell by 1.07. The authors call the changes in peak and threshold capacity medium-to-large within-subject effects.

The gains were not limited to a few people. According to the study, 82.9% of patients showed some increase in VO2 max per kilogram. In 53.9% it rose by at least 0.5 MET, a unit of exercise intensity, and in 26.6% by at least 1 MET. Peak or threshold oxygen uptake improved in 94.4%. Gains in aerobic capacity appeared across clinical indications, age groups from under 45 to 75

and older, and in both sexes.

The authors place the numbers in context. Their introduction cites a cohort of 122,007 adults in which higher fitness was associated with lower all-cause mortality, with no upper limit to the benefit. It also cites work associating each 1-MET increase in fitness with a 13% to 15% lower relative risk of death.

The authors state that, given the retrospective, uncontrolled design, prospective controlled studies are required to determine HBOT's specific contribution to the changes.

The study did not measure mortality or clinical events, so the fitness changes cannot establish a longevity benefit. Adverse events were not systematically recorded. One author disclosed a medical-advisory role and shareholding in AVIV Scientific.

The next step to watch is prospective controlled trials, which would show how much of the fitness change belongs to the therapy itself.

Sources

Source 1: https://www.frontiersin.org/journals/aging/articles/10.3389/fragi.2026.1962894/full

Read this article online

08 / LONGEVITY & HEALTHSPAN

Model Links Harsher Breast Cancer Chemotherapy to Earlier Aging-Related Disease

A computer model projects earlier aging-related mortality after more cytotoxic breast cancer chemotherapy. Its life-year estimates are simulated outcomes, not losses observed in patients.

The finding comes from a computer simulation, not from tracking patients. Published in JAMA Network Open in 2026, the study used an established breast cancer model from the Cancer Intervention and Surveillance Modeling Network. It simulated 400 million US women diagnosed with breast cancer between ages 30 and 79, with diagnoses between 1990 and 2030 and follow-up until death.

The model tracked biological aging, meaning a body that is older than its birth date suggests. It modeled this using data on p16INK4a, a marker of senescent cells. These are cells that have stopped dividing but resist dying. The model assumed that chemotherapy-driven senescence affected only deaths from causes other than breast cancer.

The researchers ran two scenarios. In one, every woman received an anthracycline. In the other, every woman received a nonanthracycline regimen. Anthracyclines are the more cytotoxic, or cell-damaging, option in this comparison.

The model projected a loss of 9.4 years of remaining life (interquartile range, 7.6 to 11.2). The risk of death from causes other than breast cancer was projected to overtake the risk of death from breast cancer 16 years earlier than expected for their chronological age (interquartile range, 12 to 19).

Comparing the two regimens, women on the more cytotoxic one were estimated to lose a median of 7.6 more years of life (interquartile range, 7.0 to 8.0). The study attributes this to aging-related diseases starting earlier. Results varied widely, and the spread was greater for women diagnosed at younger ages than at older ages.

The study also reported a trade-off. Survival from breast cancer was higher with the more cytotoxic regimens. That benefit came paired with a greater potential risk of earlier death from other causes because of age acceleration. The model closely reproduced all-cause survival and 8-year breast cancer survival seen in the Anthracycline for Early Breast Cancer trials.

For survivors and their doctors, the authors say the value lies in awareness and in survivorship care. They write that integrating knowledge about biological aging into that care could inform future research on accelerated aging among survivors. According to GEN, which covered the study, the researchers said oncologists should not change treatment decisions because of possible accelerated aging, since the goal of treatment is to improve breast cancer survival.

The authors add that as evidence on senolytic and other anti-aging drugs develops, modeling may help identify survivors who could benefit. These potential uses still need clinical testing.

Next to watch is research testing whether measuring and managing biological age improves cancer survivors' outcomes. The simulation alone does not establish how an individual's cancer treatment should change.

Sources

Source 1: https://jamanetwork.com/journals/jamanetworkopen/fullarticle/2856363

Source 2: https://doi.org/10.1001/jamanetworkopen.2026.43008

Source 3: https://lombardi.georgetown.edu/news-release/chemotherapy-for-breast-cancer-linked-to-accelerated-biological-aging/

Read this article online

09 / LONGEVITY & HEALTHSPAN

Triglyceride-Rich Particles and Lp(a) Share Blood Protein Signals of Artery Inflammation

LDL cholesterol is the number most people hear about at a heart check. A large study in the European Heart Journal suggests it may not tell the whole story: two other kinds of fat-carrying particles carry blood protein signals of inflammation that LDL does not.

The work is an observational study in people. Researchers led by Jakub Morze and Clemens Wittenbecher at Chalmers University of Technology in Sweden analysed 35,269 UK Biobank participants who had no known heart disease, stroke or peripheral artery disease at the start. They checked their findings in a second group, the Multi-Ethnic Study of Atherosclerosis, with 5,915 participants.

Three kinds of particle carry fats through the blood and all share a surface protein called apolipoprotein B: LDL, triglyceride-rich lipoproteins (TRL) and lipoprotein(a), or Lp(a). All three can lodge in artery walls. The authors note that, per particle, TRL are three to four times and Lp(a) four to five times more strongly associated with coronary artery disease than LDL, according to earlier research.

To look for reasons, the team measured 2,918 blood proteins. They linked proteins to each particle type using two approaches: standard regression and Mendelian randomization, a genetic method. Only proteins found by both were kept. That left 30 proteins tied to LDL, 471 to TRL and 53 to Lp(a).

The TRL and Lp(a) protein patterns were distinct from the LDL pattern but overlapped with each other. They shared 36 proteins, and these were enriched in inflammatory pathways.

The patterns also tracked with later disease. Over follow-up in UK Biobank, 1,599 people had a coronary event. The authors combined the proteins into a score for each particle type and adjusted for other risk factors and for measured lipoprotein levels. Each one-standard-deviation rise in the TRL score was associated with a hazard ratio of 1.16 for coronary artery disease. For Lp(a) it was 1.09 (95% confidence interval 1.03 to 1.15). For LDL it was 0.95, with a confidence interval of 0.89 to 1.02, which shows no clear link.

The authors also estimated how much of each particle's risk ran through these protein changes. Markers of immune activation and vascular remodelling, meaning changes in the structure of blood vessel walls, were consistent with explaining 62% of the TRL-associated risk and 14% of the Lp(a)-associated risk. The authors conclude that, beyond depositing fat in arteries, TRL and Lp(a) converge on inflammatory and vascular remodelling pathways that may explain their greater per-particle harm compared with LDL. That mediation estimate rests on observational assumptions.

The study did not test a treatment strategy for patients with low LDL. Its findings concern protein signatures and later coronary risk, not a demonstrated improvement in clinical care.

The finding concerns protein scores after adjustment, not evidence that LDL itself is harmless or that lowering LDL is unimportant.

For readers, the practical point is about what a standard LDL result captures. LDL-related proteins in this study mostly were not tied to later coronary disease once other measures were accounted for. Triglyceride-rich particles and Lp(a) were. Those are the measures that sit alongside LDL in the paper's account of artery risk.

Next to watch is whether trials targeting TRL or Lp(a) show that lowering them also changes these inflammatory protein signals and reduces heart events.

Sources

Source 1: https://pubmed.ncbi.nlm.nih.gov/42671122/

Source 2: https://doi.org/10.1093/eurheartj/ehag718

Read this article online

10 / LONGEVITY & HEALTHSPAN

Vesicles Shed by Mitochondria Carry Microglial Protein Ggt1 Into Alzheimer's Pathology

Tiny packages shed by the brain's mitochondria may help carry Alzheimer's disease from cell to cell, according to a study in Nature Aging. The packages, called mitovesicles, were more abundant in Alzheimer's brains, and they carried a protein called Ggt1 that the researchers tie to the damage.

The work is laboratory research. It used brain tissue from patients with Alzheimer's disease and 5×FAD mice, a standard mouse model of the disease, along with wild-type mice. No treatment has been tested in people.

Mitochondria are the energy-producing structures inside cells. Cells can release mitochondrial components inside extracellular vesicles, small membrane-wrapped packets. Mitovesicles are a recently identified type.

The team found that release of mitovesicles was enriched in brain tissue from patients with Alzheimer's and in 5×FAD mice.

The key experiments tested whether the vesicles could drive disease. Mitovesicles from 5×FAD mouse brains, which the authors call ADmtVs, were administered to wild-type mice. They induced Alzheimer's-like behavioral and pathological changes. The same vesicles accelerated disease progression in mice that already had the Alzheimer's model.

The researchers also examined what controls release. According to the paper, mitovesicle release was regulated by mitochondrial respiratory metabolism. Blocking release significantly alleviated Alzheimer's-like features in the mice.

Proteomic analysis, a survey of the proteins in the vesicles, identified Ggt1 as highly enriched in ADmtVs. Ggt1 is an enzyme that breaks down glutathione, an antioxidant that helps cells manage oxidative stress. Glutathione levels are reduced in the brains of Alzheimer's patients and animals, the authors note. The study describes Ggt1 as a microglial protein, and microglia are the brain's immune cells.

Reducing Ggt1 helped. Inhibiting its expression and activity mitigated Alzheimer's-like features in 5×FAD mice and delayed the disease progression induced by ADmtVs. The authors suggest Ggt1 is a pathological cargo of the vesicles.

For people thinking about brain aging, the finding offers a proposed route linking mitochondrial stress, immune cells and the spread of pathology. The authors say mitovesicles and Ggt1 are potential therapeutic targets. The evidence so far comes from mice and human tissue, so whether the same chain operates in living patients is unknown.

Next to watch is whether other groups reproduce the findings and whether interventions aimed at Ggt1 or vesicle release advance beyond mouse models.

Sources

Source 1: https://pubmed.ncbi.nlm.nih.gov/43009615/

Source 2: https://doi.org/10.1038/s43587-026-01263-3

Read this article online

11 / LONGEVITY & HEALTHSPAN

Implanted Vagus Nerve Stimulator Linked to Sustained Responses in Rheumatoid Arthritis at Two Years

A small device wrapped around a nerve in the neck, switched on for one minute a day, produced more improvement in rheumatoid arthritis than a sham device in a randomized trial, and most patients were still using it two years later.

The trial, called RESET-RA, was published in Nature Medicine in 2026. It was a double-blind, sham-controlled randomized trial of 242 adults with moderately to severely active rheumatoid arthritis. All had failed to respond to, or could not tolerate, at least one biologic or targeted synthetic disease-modifying drug. These are the advanced drugs used after standard treatments such as methotrexate.

The idea rests on what the authors call the inflammatory reflex. The vagus nerve carries signals between the brain and the body's organs. In this reflex, vagus nerve activity prompts immune cells to release less of the chemical messengers that drive inflammation, including TNF, IL-6 and IL-1β. The paper reports that in rheumatoid arthritis this nerve activity is diminished, and that the drop precedes the onset of clinical disease.

The implant is placed on the left vagus nerve in the neck. Active stimulation was set to the patient's upper comfort level, up to 2.5 mA, and delivered a one-minute train of pulses once daily at 10 Hz. Patients had lived with the disease for an average of 12.4 years. They had tried a median of two advanced drugs before enrolling.

After surgery, patients were randomized to active or sham stimulation for three months. The primary measure was ACR20, a standard score meaning at least a 20% improvement in tender and swollen joints and in several other measures, such as pain and disability. ACR20 was reached by 35.2% of the active group and 24.2% of the sham group (one-sided $P = 0.0209$). The adjusted difference was 11.8 percentage points, with a confidence interval of 0.6 to 23.1.

A secondary measure showed a larger gap. A good or moderate response by European rheumatology criteria occurred in 60.7% with stimulation and 41.7% with sham. Other secondary measures also favored stimulation, but did not reach statistical significance after adjustment for multiple comparisons.

After three months, everyone received stimulation in an open-label phase. ACR20 rates in the original stimulation group rose to 50.0% at six months and 52.8% at 12 months among all completers. Across both groups at 12 months, 57.6% reached ACR20 in an analysis that excluded patients given added drug therapy. During the open-label year, 32.2% of patients received added drug treatment by month 12, and 75.2% remained free of added biologic or targeted drugs.

On safety, adverse events occurred in a similar share of patients in both arms. Related serious adverse events occurred in 1.6% of patients. All happened around the time of surgery, and all

resolved.

Longer follow-up was presented at the American College of Rheumatology's Convergence 2026 meeting, according to SetPoint Medical’s conference announcement. John Tesser, the trial's national rheumatology principal investigator, reported the 24-month results. Among patients not taking a concomitant biologic or targeted drug, 62.7% reached ACR20, 38.7% reached ACR50 and 18.7% reached ACR70. About half reached low disease activity or remission. At 24 months, 91.7% were still using stimulation and 29.3% were also taking an advanced drug. Fourteen patients (5.8%) had the device removed, most often because of perceived lack of effectiveness. The company reported no serious adverse events attributed to the device or stimulation from month three to month 24.

The 24-month figures come from open-label follow-up with no sham group, so only the three-month comparison isolates the effect of stimulation.

The US Food and Drug Administration approved the SetPoint System in July 2025 for adults with moderately to severely active rheumatoid arthritis after inadequate response, loss of response or intolerance to at least one biologic or targeted synthetic disease-modifying drug, according to the company. What to watch next is whether longer controlled comparisons confirm that the benefits seen in open-label use hold up against sham, and how the device fits alongside biologic and JAK inhibitor drugs.

Sources

Source 1: https://pubmed.ncbi.nlm.nih.gov/41429981/

Source 2: https://pmc.ncbi.nlm.nih.gov/articles/PMC12823386/

Source 3: https://www.prnewswire.com/news-releases/setpoint-medical-presents-24-month-data-demonstrating-sustained-clinical-benefit-and-favorable-long-term-safety-of-the-setpoint-system-for-rheumatoid-arthritis-302932955.html

Read this article online

12 / LONGEVITY & HEALTHSPAN

Beam's Base-Edited Sickle Cell Therapy Meets Main Goal, FDA Filing Planned

Beam says its investigational base-edited cell therapy met the primary endpoint in a pivotal Phase 1/2 trial. The company plans an FDA submission as early as the end of 2026.

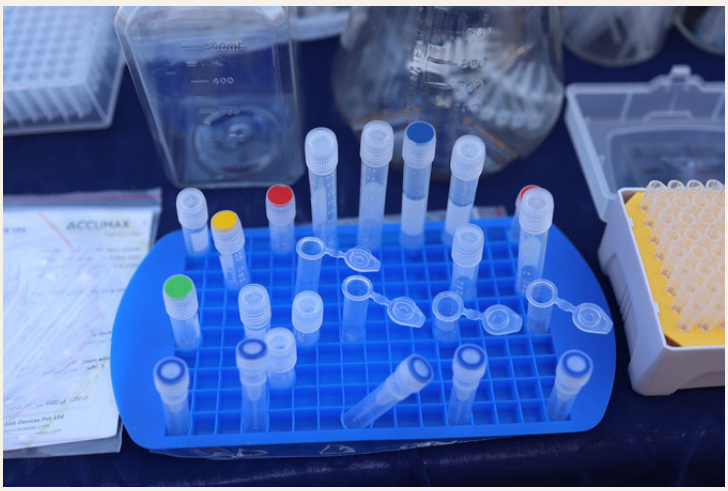


Photo: “Laboratory microcentrifuge tubes and pipette tips 01” by Gannu03, CC BY-SA 4.0

Beam Therapeutics said Tuesday that its treatment, ristoglogene autogetemcel, or risto-cel, met the main goal of its pivotal trial: freedom from severe vaso-occlusive crises. These are the painful episodes that sickle cell disease causes. According to the company, the treatment also met secondary goals, including freedom from inpatient hospitalization for severe crises for at least 12 consecutive months. As of October 20, 49 participants had been treated, and 31 had completed the required follow-up for the primary endpoint.

The approach is called base editing. It can change individual DNA letters precisely and, unlike older CRISPR methods, does not make double-stranded DNA breaks. Doctors take a patient's own cells and re-engineer them to switch on production of fetal hemoglobin, a special oxygen-carrying protein. The cells go back into the patient, settle in the bone marrow and start making normal blood cells. The aim is to stop sickle-shaped cells from forming and causing crises.

Beam says the planned filing could be the first biologics license application for a base-editing therapy. The trial is open-label and single-arm, and the announcement does not provide the detailed pivotal response rate.

Treatment requires collection of the patient’s own blood-forming stem cells, editing outside the body, and a transplant procedure with myeloablative busulfan conditioning. The company said safety findings were consistent with the known risks of these procedures and the underlying disease.

Next to watch are the detailed pivotal results, planned for a medical meeting in 2027, and whether Beam submits its FDA application as early as year-end 2026.

Sources

Source 1: https://investors.beamtx.com/news-releases/news-release-details/beam-therapeutics-announces-positive-topline-pivotal-data-beacon

Read this article online

13 / LONGEVITY & HEALTHSPAN

Home Exercise Improved Cancer Survivors' Peak Oxygen Uptake, Heart Protection Unproven

Structured exercise programmes done at home improved the fitness of people who had received cancer treatments that can harm the heart, but whether they protect the heart itself is still unproven.



Photo: by Shixart1985, CC BY 2.0

That is the conclusion of a systematic review and meta-analysis published in Supportive Care in Cancer in 2026. The authors, Wang, Lee, Zhang and Liu, pooled results from 10 randomised controlled trials involving 851 participants. All were cancer survivors who had received cardiotoxic therapies. The team searched seven electronic databases for trials published from January 2000 to October 2025.

The main fitness measure was peak oxygen uptake, known as VO2 peak. It reflects how much oxygen the body can use during hard exercise and is a standard marker of cardiorespiratory fitness. According to the meta-analysis, home-based exercise significantly improved VO2 peak by an average of 2.35 mL/kg/min (95% confidence interval 1.26 to 3.43, $p < 0.001$).

The size of the gain varied. Subgroup analyses found larger effects in programmes lasting 24 to 52 weeks, in programmes that used mobile health (mHealth) tools, and in people with stage III or IV cancer.

Other outcomes were less clear-cut. The pooled results showed no significant improvement in the six-minute walk test, a measure of functional capacity, or in health-related quality of life. Even so, the authors concluded that home exercise mitigated functional decline in cancer survivors.

The central question for the heart is still open. The review set out to look at direct measures of heart damage, such as left ventricular ejection fraction and global longitudinal strain, and at blood markers such as troponins and B-type natriuretic peptide. It also looked at clinical events

such as heart failure and arrhythmias. Only one of the 10 trials assessed the direct cardiotoxicity measures and biomarkers.

The authors therefore write that the ability of home-based exercise to prevent cancer treatment-related cardiovascular toxicity was unproven. A higher VO2 peak is a sign of better fitness. In this analysis it was not shown to mean less damage to the heart from treatment.

For survivors, the practical picture from this evidence is a measurable fitness gain from exercise done at home. Whether it also shields the heart remains to be tested.

The authors call for adequately powered trials that include direct cardiotoxicity assessments to establish true cardioprotective efficacy. They also call for implementation studies to optimise how home programmes are delivered in the real world.

Sources

Source 1: https://pubmed.ncbi.nlm.nih.gov/42975689/

Source 2: https://doi.org/10.1007/s00520-026-11434-y

Read this article online

14 / LONGEVITY & HEALTHSPAN

Company Reports Zabopegdutide Resolved MASH in 63% in Phase 2 Biopsy Data

Company-reported liver-biopsy findings suggest benefit in a small per-protocol subset. Larger studies are needed to establish efficacy and safety.

D&D Pharmatech has reported 48-week Phase 2 liver-biopsy results for zabopegdutide. The central response rates were first disclosed in May 2026; they are company-reported findings, not a new November readout.

The conventional biopsy percentages come from a per-protocol group of 35 participants with paired biopsies: 16 on zabopegdutide and 19 on placebo, out of 67 enrolled. The qFibrosis analysis involved a different subset of 27: 11 treated and 16 placebo. These small selected subsets limit certainty and generalizability.

MASH is fatty liver with active inflammation and liver cell damage. Per the report, 62.5% of the treated group reached MASH resolution without worsening fibrosis, meaning scarring, versus 5.3% on placebo ($p<0.001$).

Scarring itself moved too. According to the report, 50.0% of the treated group had fibrosis improvement without worsening MASH, versus 15.8% on placebo ($p=0.03$). A composite response, a combined measure, was reached by 37.5% versus 5.3% ($p=0.02$).

The company also claims that digital pathology, an AI-based reading of biopsy slides using a tool called qFibrosis, found at least one stage of fibrosis improvement in 81.8% of treated patients. The continuous score fell 41.0% from baseline with treatment and rose 9.5% with placebo, a placebo-adjusted difference of 50.5 percentage points.

According to the company, noninvasive liver-fat, liver-stiffness and metabolic measures also improved. The most common adverse events were gastrointestinal and generally mild to moderate; dosing began with a two-week titration.

Biopsy results matter because they look at the liver tissue directly rather than at fat levels alone.

Next to watch are larger trials and fuller safety and efficacy reporting. The drug remains investigational.

Sources

Source 1: https://ddpharmatech.com/en/board/board.php?bo_table=press_en&idx=20

Source 2: https://ddpharmatech.com/board/board.php?bo_table=press&idx=96

Read this article online

15 / LONGEVITY & HEALTHSPAN

Late Chronotype Linked to Metabolic Risk and Gut-Barrier Differences

An observational analysis of 87 adults with obesity linked late chronotype with greater metabolic syndrome burden. Gut permeability and inflammatory markers also varied with circadian habits; the study does not establish causation.



Photo: by PattayaPatrol, CC BY-SA 4.0

The work is an observational analysis of participants drawn from a randomized clinical trial. All 87 adults had a body mass index above 30. Their mean age was 42.5 years. The team, led by Faraz Bishehsari at UTHealth Houston, looked at two body-clock habits: chronotype, meaning whether a person naturally runs early or late, and how close to sleep time they ate.

Metabolic syndrome is a cluster of problems that includes obesity, insulin resistance, abnormal blood fats and high blood pressure. It raises the risk of cardiovascular disease, stroke and type 2 diabetes. The researchers split participants into a lower-risk group with zero or one feature and a higher-risk group with two or more.

Late chronotype was common: 59 of 87 participants (67.8%) were night owls. Of these, 42 (71.2%) were in the higher-risk group, compared with 13 of 28 early types (46.4%). Late eating was even more common. Some 66 participants (75.9%) were classed as late eaters, and 39 of them (59.1%) were in the higher-risk group.

The two habits together pointed the same way. The share in the higher-risk group rose from 6 of 14 (42.9%) among early types who ate early to 16 of 22 (72.7%) among late types who ate late. That combined-phenotype trend did not reach conventional statistical significance ($p = 0.07$).

To look beneath the clinical labels, the team built a composite metabolic-inflammatory score from 15 blood measures, including cytokines, metabolites and sugar-chain structures on antibodies. The score separated the two risk groups well in within-cohort testing, with an area under the curve of 0.918. Higher scores were tied to older age and poorer diet quality.

Specific markers lined up with the habits. Late chronotype was linked to higher interferon gamma, an inflammatory signal, and lower levels of the amino acid tryptophan. Late eating was linked to higher levels of the immune-signalling chemokine CCL8 and to a distinct antibody sugar profile.

The gut barrier is where the story turns. Gut permeability, measured with a sugar absorption test, was higher in the higher-risk group. Circadian misalignment was associated with increased permeability and inflammation, and the link was stronger in participants whose guts were already more permeable. Beta-glucan, a marker of fungal material crossing the gut wall, was higher in the higher-risk group.

These patterns went with lower levels of bacteria associated with short-chain fatty acids, compounds gut microbes make that support the gut lining. In an exploratory analysis, participants whose permeability changed after a microbiome-targeted intervention showed matching changes in these bacteria.

The main analyses used baseline measurements, so they cannot say whether late schedules led to the gut and inflammation findings. Medical Xpress framed the results as showing how late habits may weaken the barrier. The paper reports associations.

Future trials will need to test whether changing circadian habits or gut-barrier function improves metabolic outcomes.

Sources

Source 1: https://pubmed.ncbi.nlm.nih.gov/42927177/

Source 2: https://pmc.ncbi.nlm.nih.gov/articles/PMC13694823/

Source 3: https://doi.org/10.1016/j.isci.2026.117892

Read this article online

16 / LONGEVITY & HEALTHSPAN

Urolithin A Supplement Failed to Improve Artery Function in Adults With Obesity

Four weeks of urolithin A, a supplement promoted for cellular health, did not significantly change how well arteries relax in middle-aged adults with obesity, according to a randomized clinical trial in JAMA Network Open.



Photo: “Omega 3 capsules in a bottle (52715343808)” by Jernej Furman from Slovenia, CC BY 2.0

The trial was double-blind and placebo-controlled. It enrolled 50 adults aged 40 to 64 with a body mass index of 30 or higher: 25 took 1000 mg of urolithin A and 25 took a placebo. The mean age was 50.7 years, mean BMI was 38.3, and 74% of participants were female. Follow-up averaged 28 days.

Urolithin A is a compound made by gut bacteria from polyphenols, plant chemicals found in some foods. The researchers describe it as an activator of mitophagy, the cell's process for clearing out damaged mitochondria, the structures that produce energy. Mitochondrial problems contribute to dysfunction of the endothelium, the lining of blood vessels, in obesity. That made the supplement a plausible candidate.

The main measure was brachial artery flow-mediated dilation, a standard test of how much an arm artery widens when blood flow increases. Mean flow-mediated dilation increased by 1.3 percentage points in the urolithin A group and 0.3 percentage points with placebo. The difference was not statistically significant. Other tests of blood vessel function, in the hand and fingertips, also showed no significant treatment effect.

The brain measures told a different story. During mental tasks, brain blood-flow responses were significantly greater after urolithin A than after placebo. The mean β coefficient, a measure of the size of the response, was 15.53 in the supplement group and −1.43 in the placebo group. Task performance, however, did not change.

The authors concluded that the supplement was well tolerated and feasible. They said its effect on task-evoked brain blood flow warrants further investigation. Seven participants, or 14%, were lost to follow-up.

The trial was registered at ClinicalTrials.gov as NCT05921266. What to watch next: whether longer studies of urolithin A, and trials focused on brain blood flow, can show if the brain signal translates into measurable benefits.

Sources

Source 1: https://pubmed.ncbi.nlm.nih.gov/42853569/

Source 2: https://doi.org/10.1001/jamanetworkopen.2026.38047

Read this article online

17 / LONGEVITY & HEALTHSPAN

Faster Everyday Walking Pace Linked to Lower Death Risk in Older Adults

How quickly someone walks down the street may say something about how long they will live. A large study of older British adults found that faster walking, measured in two different ways, was linked to a lower risk of death over nearly 12 years.

The research, published in JAMA Network Open in 2026, is an observational study in people. It drew on the Whitehall II cohort in the UK and included 4,067 participants with an average age of 69.3. About three in four were men. Baseline measurements were taken in 2012 and 2013, and deaths were tracked through August 2024 using the British national mortality register.

Walking pace was captured in two ways. First, a nurse timed each participant over a 2.44-metre course. They were asked to walk at their usual pace, as if walking down the street to the shops. The test was done three times and the average was used. Second, participants wore an accelerometer on the wrist for nine days. From that data, researchers worked out walking cadence: the number of steps taken per minute of walking.

The researchers used two cadence measures. One was the median cadence, a typical walking pace. The other was the 95th percentile cadence, which reflects the fastest walking someone does in daily life. Participants averaged 75.3 steps per minute at the median and 112.2 steps per minute at the 95th percentile. Average clinical walking speed was 1.2 metres per second.

During a median follow-up of 11.8 years, 739 participants (18.2%) died. After accounting for age, sex, education, ethnicity, marital status, smoking, alcohol, fruit and vegetable intake, body mass index, blood pressure, cholesterol, daily living limitations and chronic disease, all three measures were linked to lower risk of death. The figures are per one standard deviation higher, a standard statistical step of difference between people.

For clinical walking speed, the hazard ratio was 0.77 (95% confidence interval 0.71 to 0.84). For median cadence it was 0.83 (0.77 to 0.90), and for 95th percentile cadence 0.82 (0.75 to 0.89). A hazard ratio below 1 means a lower rate of death. The patterns were similar in women and men.

The three measures also performed about equally well at telling apart people who died from those who did not. The study used a score called the C index for this, where 0.5 is chance and 1 is perfect. Clinical walking speed scored 0.747, median cadence 0.744 and 95th percentile cadence 0.745. Only clinical walking speed improved the discriminative ability of a model that already included the standard risk factors.

The two kinds of measurement were not closely tied together. Correlations between the clinic test and the wrist-based cadence measures were weak, at 0.28 for median cadence and 0.27 for 95th percentile cadence. The authors conclude that both can be used in population studies, and that the choice may depend on research goals and practical constraints.

The study tracked who walked faster and who died, and did not test whether speeding up changes lifespan. Its sample was also healthier than the wider group of Whitehall II participants who took part in the 2012 to 2013 wave of data collection.

Sources

Source 1: https://pubmed.ncbi.nlm.nih.gov/42912394/

Source 2: https://pmc.ncbi.nlm.nih.gov/articles/PMC13695056/

Source 3: https://doi.org/10.1001/jamanetworkopen.2026.40122

Read this article online

18 / LONGEVITY & HEALTHSPAN

UK Launches £20 Million AI Hub for Earlier Dementia Diagnosis

Dementia is often diagnosed late, and about one-third of people living with it in the UK have never had a formal diagnosis. A new £20 million research hub plans to build artificial intelligence tools inside NHS clinics to find the earliest signs of the disease.

The hub is called BRAINIAC. According to Newcastle University, it is funded with £10 million from the Engineering and Physical Sciences Research Council, part of UK Research and Innovation. Industry, charity and NHS partners will add a further £10 million. The University of Exeter leads it, with researchers from 11 universities and 10 NHS sites across all four UK nations.

The need is large. Around 982,000 people in the UK live with dementia, a figure projected to reach 1.4 million by 2040. Annual costs are expected to rise to more than £90 billion over the same period. Many people also face long waits for specialist assessment.

BRAINIAC will develop AI systems that combine several kinds of information into one picture of a person's brain health. These include blood biomarkers, brain imaging, genetics, electronic health records and digital cognitive assessments. Blood tests can now detect signs of Alzheimer's disease years before symptoms appear, according to the university. The AI tools will be built and tested directly in NHS brain health clinics.

Over six years, the hub will support 10 new brain health clinics. The first are expected to open in 2029 and will initially be reached through NHS referral. The hub will work with more than 1,700 patients. Its aims are to cut assessment and diagnosis times by 30–50 per cent and to identify more than 40 per cent of people at risk before symptoms develop. It also plans to let people complete assessments at home through online platforms and apps. These figures are goals set by the project, not results.

The clinics will be linked through secure systems that let hospitals share AI models without sharing patient data. The hub also aims to create one of the UK's largest trial-ready groups of patients for dementia research.

Professor David Llewellyn, the hub's director, said the tools "will let us pick up the first signs before symptoms show and offer people a personalised prevention plan." Professor Chris Fox, deputy director and a practising clinician, said: "The treatments now arriving only help if we find people early." Dr Judith Harrison of Newcastle University said the aim is to give people more time to make informed decisions about their health.

People living with dementia and carers will help design the technologies, working with Innovations in Dementia. Alzheimer's Society will support efforts to bring successful tools into NHS practice. The hub will also train more than 200 clinicians and engineers and support between 10 and 20 small companies.

Next to watch are the first BRAINIAC clinics, expected from 2029, and whether the project meets its diagnostic targets.

Sources

Source 1: https://www.ncl.ac.uk/press/articles/latest/2026/11/brainiac/

Read this article online

19 / LONGEVITY & HEALTHSPAN

Review Links Gut Bacteria to Parkinson's Inflammation and Levodopa Availability

Constipation that shows up a decade or more before tremor or stiffness may be an early clue to Parkinson's disease in some people, and gut bacteria may also change how the main Parkinson's drug behaves in the body, according to a new review in Frontiers in Immunology.

The paper, published Nov. 10 by researchers at Nova Southeastern University and other institutions, is a review. It gathers existing evidence from mouse experiments, human cohort studies and early trials rather than reporting new data. Parkinson's affects more than 10 million people worldwide, the authors write.

The central idea is a "gut-first" form of the disease. In a subset of patients, the authors write, the misfolded protein α-synuclein may begin to build up in the nerves of the gut and then travel to the brain. In transgenic mice, α-synuclein overproduction in gut nerves led to clumps there, spread to the brainstem along the vagus nerve, and caused slowed gut movement. In another mouse study the review describes, injecting α-synuclein fibrils into the duodenum led to spread toward the substantia nigra, the brain region that loses dopamine-producing cells in Parkinson's. Cutting the vagus nerve blocked that spread.

The gut-first account remains a proposed subtype model; the review notes that human autopsy findings neither establish nor exclude it.

A human association study provides indirect support, but does not establish gut-to-brain causation. In a prospective cohort followed for 24 years, fewer than one bowel movement a day was linked to a 4.1 to 4.5 times higher risk of Parkinson's, with an average of about 12 years between constipation and the start of movement symptoms. The authors also name REM sleep behavior disorder and cardiac sympathetic denervation as signs that may mark the gut-first subtype.

The review lays out how gut bacteria may feed brain inflammation. In dysbiosis, an imbalanced gut community, bacteria that make short-chain fatty acids decline, bacteria that make lipopolysaccharide increase, and the gut barrier weakens. According to the authors, this activates a signaling pathway called TLR4/NF-κB, primes microglia (the brain's immune cells) toward a disease-linked state, and promotes α-synuclein misfolding. They add that the complement system, part of the immune response, links gut bacteria to nerve damage.

Medication is the second theme. The authors report that gut bacteria influence how levodopa moves through the body via bacterial tyrosine decarboxylase activity. The microbiome also interacts with COMT inhibitors, drugs often used alongside levodopa, and alters how much drug becomes available.

The review also covers possible markers for diagnosis. These include the ratio of trimethylamine N-oxide to butyrate, secondary bile acids, phosphorylated α-synuclein in skin, and seeding amplification assays, which detect misfolded protein. The authors cite epidemiological evidence

linking earlier antibiotic exposure and inflammatory bowel disease to Parkinson's risk.

Treatments aimed at the gut are in early testing. The review covers short-chain fatty acid supplements, fecal microbiota transplantation, GLP-1 receptor agonists and targeted probiotics. According to the authors, microbiome-directed approaches may help most in the gut-first subtype, which is why they call for trials that sort patients by subtype.

Next to watch: trials that enroll patients by gut-first features such as prodromal constipation or REM sleep behavior disorder, which will show whether microbiome-directed therapies work in the people the theory predicts.

Sources

Source 1: https://www.frontiersin.org/journals/immunology/articles/10.3389/fimmu.2026.1975641/full

Read this article online

20 / LONGEVITY & HEALTHSPAN

Sironax to Show Preclinical Data on NAD-Pathway Obesity Pill

Weight-loss drugs can shrink fat, but people often lose muscle along with it. According to its November 4 announcement, a company called Sironax claims that an experimental pill aimed at a cellular energy pathway produced preferential fat loss in preclinical findings.



Photo: “Capsules” by Photo taken byAney, CC BY-SA 3.0

According to Sironax's November 4 announcement, Sironax will present two posters on the drug, SIR4156, at ObesityWeek 2026. The Obesity Society's annual meeting runs Nov. 14–17 at the Gaylord National Resort and Convention Center in National Harbor, Maryland. Both posters are scheduled for Nov. 14, from 7:30 to 8:30 p.m. ET.

SIR4156 is described as a first-in-class, orally active allosteric activator of NAMPT. In plain terms, it is a pill meant to switch on an enzyme. NAMPT is part of the pathway that cells use to make NAD+, a molecule involved in producing energy.

One poster is titled Pharmacologic NAMPT Activation Improves Metabolism with Weight-Lowering Effects in DIO Mice. DIO stands for diet-induced obesity, meaning mice made obese through diet. The other poster is a weight-of-evidence carcinogenicity risk assessment of SIR4156 in obesity, which looks at cancer risk.

According to the report, the company claims its preclinical findings include preferential fat loss, lean-mass preservation, reduced muscle atrophy and improved physical performance. These are the company's own claims, from animal work.

Why this matters for people using GLP-1 drugs: weight lost on these medicines can include lean mass, which includes muscle. A drug that spared muscle could change what a good weight-loss result looks like. According to the report, Sironax is developing SIR4156 both on its own and in combination with incretins, the class that includes GLP-1 drugs.

The report gives no human efficacy data. It says Sironax has advanced enrollment in its Phase 2 FORGE-LM obesity trial of SIR4156. Results from mice have often failed to carry over to people, so the human trial is where the question gets tested.

Watch for what the posters show on Nov. 14, and for any results from the FORGE-LM trial.

SIR4156 remains investigational; its safety and efficacy in humans have not been established.

Sources

Source 1: https://www.globenewswire.com/news-release/2026/11/04/3395149/0/en/sironax-announces-two-abstracts-on-nampt-activator-sir4156-selected-for-presentation-at-obesityweek-2026.html

Read this article online

21 / LONGEVITY & HEALTHSPAN

Alpha-1 Liver Disease Gets a Shared Definition as RNA Editing Data Emerge

Liver disease caused by alpha-1 antitrypsin deficiency (AATD) is still going unrecognized in adults, and a new expert consensus aims to change that by agreeing on what it is, how to stage it and how to measure whether a treatment works.

The document is a consensus guidance, not a clinical trial. Published in Gastroenterology in 2026, it comes from a multidisciplinary international panel convened by the American Gastroenterological Association with the American Association for the Study of Liver Diseases, the European Association for the Study of the Liver and the Alpha-1 Foundation. The panel combined recent epidemiologic, tissue-based and noninvasive biomarker studies with expert opinion. The lead author is Rohit Loomba and the senior author is Pavel Strnad.

According to the guidance, AATD-associated liver disease remains underrecognized despite contributing to illness and death in adults with the PiZZ genotype, the form of the inherited condition most linked to liver harm. The authors say the lack of standard definitions, diagnostic criteria and staging has slowed both diagnosis and drug development.

The panel defines the disease by liver enzyme elevations, which may come and go, and/or liver fibrosis of stage F2 or higher in adults with AATD, particularly those with PiZZ. The enzymes named are aspartate aminotransferase, alanine aminotransferase and γ-glutamyl transferase. Fibrosis is scarring of the liver.

For staging, the panel preferred noninvasive elastography, which measures liver stiffness, over other methods. It named vibration-controlled transient elastography and magnetic resonance elastography as examples. A stiffness of 8 kPa or more on the first of these was set as the threshold for clinically significant fibrosis.

An investigational treatment is WVE-006, an investigational RNA editing drug given by injection under the skin. Wave reported interim open-label phase 1b/2a results from the RestorAATion-2 trial in May 2026, in adults with PiZZ AATD, with 8 participants in each dose group. The drug edits the messenger RNA in liver cells so that the body makes normal M-type AAT instead of the faulty Z type.

According to Wave’s May 18 announcement, single doses of 200, 400 and 600 mg produced M-AAT at 44%, 48% and 52% of total AAT, with Z-AAT falling by 47%, 50% and 59%. With repeated dosing, M-AAT reached 64% and 59% of total AAT in the 200 mg and 400 mg groups, and Z-AAT fell by 71% and 68%. In the available interim data, the company reported mild-to-moderate adverse events and no serious events.

The company had already reported these figures in May 2026. They are mean maximum biomarker responses, not demonstrated improvements in clinical liver or lung outcomes. The data are interim and come from small dose cohorts.

Next to watch: whether larger trials of RNA editing use the new liver definitions and stiffness thresholds as endpoints, and how the full RestorAATion-2 results compare with the interim figures.

Sources

Source 1: https://pubmed.ncbi.nlm.nih.gov/41390004/

Source 2: https://doi.org/10.1053/j.gastro.2025.12.012

Source 3: https://www.globenewswire.com/news-release/2026/05/18/3297034/0/en/wave-life-sciences-announces-positive-update-on-restoraation-2-trial-wve-006-galnac-rna-editing-achieves-mz-like-phenotype-across-both-biweekly-and-monthly-dosing.html

Read this article online

22 / LONGEVITY & HEALTHSPAN

Oura Agrees to Acquire AI Care Startup Counsel Health

Oura announced a definitive agreement on November 10 to acquire Counsel Health, with closing expected near the end of the year.

Counsel Health makes a chatbot that people can talk with about a health concern. After that conversation, the user is transferred to a clinician for diagnosis and treatment over telehealth.

For people who wear a ring or other tracker, the deal points to a changed role for the device. Oura has already partnered with Counsel since May 2026; the agreement would bring the service under the same ownership. Under one owner, the same company would run both the data and a route to a clinician.

The agreement would connect wearable-device information more closely with clinician-supervised digital care.

The announcement is a business transaction, and it does not show whether chatbot-guided care leads to better health outcomes.

Next to watch is whether the acquisition closes and how Oura integrates Counsel’s care services.

Sources

Source 1: https://ouraring.com/blog/oura-to-acquire-counsel-health/

Read this article online

23 / LONGEVITY & HEALTHSPAN

Humacyte Begins First Human Trial of Lab-Grown Coronary Bypass Vessel

Humacyte has opened enrollment for a first human study of a lab-grown coronary bypass vessel. Human safety and efficacy results are not yet available.

According to Humacyte's November 9 announcement, Humacyte has opened enrollment in a Phase 2a clinical study of its investigational coronary tissue-engineered vessel, known as CTEV. The study involves patients undergoing coronary artery bypass grafting, a surgery that reroutes blood around blocked heart arteries.

The report says the FDA accepted Humacyte's investigational new drug application in July 2026. The trial will evaluate the safety and efficacy of the CTEV as a vascular conduit, meaning a channel that carries blood, in ten adult patients. Patency, a measure of whether the graft stays open, will be assessed at two months. Follow-up will run for up to three years.

The company describes the CTEV as an off-the-shelf, small-diameter conduit designed to be repopulated by a patient's own cells. According to the report, it is made with the same manufacturing system as Humacyte's FDA-approved acellular tissue-engineered vessel.

The CTEV and Humacyte's regenerative platform were highlighted at the American Heart Association Scientific Sessions 2026 in Chicago. Presentations covered the Phase 2a study design and engineering approaches meant to speed vascular regeneration, according to the report.

Animal data were also presented. In sheep, CTEV bypasses of about 12 centimeters were implanted for up to three months, and the report says they stayed open and were populated by the host's own cells. Porcine data were also part of the preclinical work. These are animal findings, and the ten-patient trial is the first test in people.

The CTEV remains investigational and has not been approved for sale by the FDA. Next to watch: the two-month patency results from the ten patients, and the longer follow-up that extends to three years.

Sources

Source 1: https://www.globenewswire.com/de/news-release/2026/11/09/3398371/0/en/humacyte-highlights-first-in-human-coronary-bypass-study-at-american-heart-association-scientific-sessions-2026.html

Read this article online

24 / LONGEVITY & HEALTHSPAN

Tai Chi and Exercise Classes Barely Moved Sleep in Older Cancer Survivors

A tai chi and qigong class and an exercise class matched to it in intensity produced only small changes in sleep among older male cancer survivors, and neither differed significantly from usual care.



Photo: “Woman doing hip exercises on a yoga mat on the floor” by Shixart1985, CC BY 2.0

The evidence comes from a secondary sleep-outcome analysis of a randomized controlled trial in Brain Medicine, led by Evelyn Arana-Chicas and colleagues. It enrolled 113 male cancer survivors aged 55 and older. Their average age was 69, and 87% had prostate cancer. Participants were randomized to tai chi and qigong, to the intensity-matched exercise class, or to usual care. Both classes met twice a week for 12 weeks, and follow-up ran through 12 months.

Participants were selected for clinically significant fatigue, not sleep disturbance.

Sleep was measured with the Pittsburgh Sleep Quality Index, a questionnaire on which lower scores mean better sleep. At three months after the program, the primary sleep-assessment time point, sleep quality did not differ significantly between groups. No pairwise comparison of global sleep quality reached significance at any time point.

Within groups, the picture was a little more detailed. The exercise group improved from baseline at the six-week midpoint (mean change −1.20), one week after the program (−0.82) and three months after it (−1.04). The tai chi and qigong group moved in the same direction, by −0.70, −0.65 and −0.59, but those changes were not statistically significant. Every change fell below the three-point threshold the authors use for a clinically meaningful difference. By 12 months, no significant effects remained.

Two other results stood out. Sleep efficiency, the share of time in bed spent asleep, improved more in the exercise group than in the tai chi and qigong group at three months ($p = 0.04$). This exploratory subscale result involved multiple comparisons and may be a chance finding. At 12 months, 62% of the tai chi and qigong group had a questionnaire score of 5 or below, the study's definition of remission of sleep disturbance. The figures were 47% in the exercise group and 43%

in the usual care group, and the differences were not significant. The authors say the trial had limited statistical power. Attendance was similar in both classes, at 78.4% for tai chi and qigong and 76.8% for exercise.

The authors conclude that gentle movement practices and conventional exercise may both offer feasible nonpharmacologic options, while calling for standardized delivery and larger trials. This trial did not demonstrate a clear benefit in overall sleep quality.

The trial found no clear difference in global sleep quality between the two classes in this group of mostly White, mostly prostate cancer survivors. Next to watch are adequately powered trials in more diverse groups, with sleep tracked by devices as well as questionnaires.

Sources

Source 1: https://pubmed.ncbi.nlm.nih.gov/42807997/

Source 2: https://pmc.ncbi.nlm.nih.gov/articles/PMC13618391/

Source 3: https://doi.org/10.61373/bm026a.0063

Read this article online

25 / LONGEVITY & HEALTHSPAN

Short-Lived mRNA Therapy Helped Mouse Hearts Repair After Heart Attack

The adult heart cannot replace the muscle cells it loses in a heart attack. A new study in mice, published in Nature Cardiovascular Research, reports that a brief burst of two proteins can get those cells dividing again and help the heart recover.

The evidence comes from mice. The researchers used transgenic mouse models and a prototype mRNA therapy. They report that Myc and cyclin T1 need to be switched on only transiently to drive heart muscle cells to proliferate. That led to repair of the heart muscle and functional recovery after a heart attack.

Myc is a protein that switches genes on and off. According to the paper, it is needed to restore tissue in several species that can regenerate. Adult mammalian heart muscle cells, by contrast, fail to divide after damage. Earlier work had shown that transgenic expression of Myc and cyclin T1 can push adult heart muscle cells to multiply and replace lost ones. The new study shows that the signal works when it is brief.

The therapy is a modified mRNA, a temporary set of instructions that cells read to make the two proteins. The authors describe it as a strategy for turning Myc and cyclin T1 co-expression into a way to regenerate the heart.

Further numbers come from the University of Cambridge, as reported by Medical Xpress. According to that coverage, mice given the therapy after a heart attack pumped about 10% more blood with each heartbeat than mice given a mock treatment. The gains were still present at 28 days and at 12 weeks, and the university reported no observed adverse effects in those mice. This does not establish long-term or human safety. The treatment is short-lived and localized, an approach designed to reduce the risk of overgrowth.

Next, according to the Cambridge team, come refinements to the treatment, then trials in larger animals and, after that, in people.

Sources

Source 1: https://pubmed.ncbi.nlm.nih.gov/43003910/

Source 2: https://doi.org/10.1038/s44161-026-00910-7

Source 3: https://www.cam.ac.uk/stories/treatment-promotes-repair-after-heart-attack

Read this article online

26 / LONGEVITY & HEALTHSPAN

Accurate AI Health Answers May Still Steer People Toward Worse Choices

AI chatbots are now judged mostly on one question: is the answer correct? A new peer-reviewed evaluation shows how well the leading systems score on that test, while a separate argument in STAT says correctness may not be enough to protect health.

The evaluation, published in Nature Medicine in 2026 by Vishwanath and colleagues, is a benchmark comparison of AI tools, not a trial in patients. It tested two specialized clinical tools, OpenEvidence and UpToDate Expert AI, against three general-purpose models: GPT-5.2, Gemini 3.1 Pro and Claude Opus 4.6. It used 500 medical-exam questions, 500 items from a benchmark called HealthBench, and 100 real queries that physicians sent to a general-purpose model in a live clinical setting at NYU Langone Health.

The general-purpose models won every stage. On the exam questions, Gemini scored 97.4%, GPT 94.2% and Claude 90.2%. OpenEvidence scored 89.6% and UpToDate 88.4%. On HealthBench, GPT scored 88.0 out of 100, against 62.6 for OpenEvidence and 61.3 for UpToDate.

The real-query test carries the most weight, according to the authors. Twelve US clinicians reviewed the answers blind, producing 1,800 ratings of correctness, completeness, safety and clarity on a 1–4 scale. Refusals were excluded from the scored comparisons, leaving 568 responses and 1,704 ratings. Gemini averaged 3.62, GPT 3.54 and Claude 3.52. OpenEvidence averaged 3.24 and UpToDate 3.17. Google's automatic AI Overview, which many people see first in search, averaged 3.27.

Tests did not detect statistically significant differences in harmful-content or hallucination rates; this does not establish equal safety. OpenEvidence scored lowest on clarity, at 2.84. UpToDate's tool declined 19% of queries, against 1–3% for most others.

The tested systems scored highly on these medical benchmarks. The authors add that knowledge and communication benchmarks may not fully capture clinical risk. These results describe the tested versions, not a permanent ranking; possible benchmark contamination and model-judge bias limit generalization.

That gap is the subject of an opinion piece in STAT's First Opinion section by Jessica Fishman and John L. Jackson Jr. of the University of Pennsylvania. They argue that an answer can be factually right and still change behavior for the worse.

The opinion describes potentially counterproductive wording, not a randomized demonstration that these particular responses changed vaccination behavior. Their example comes from a study they describe, conducted with the Vaccine Education Center at Children's Hospital of Philadelphia. ChatGPT, Gemini and Claude were asked about chickenpox parties. According to the authors, the answers were highly accurate and ended by recommending vaccination. But all three opened by calling the parties "popular" or "common," and the authors say research shows that describing a behavior as common can encourage it. They also say ChatGPT's description of

vaccination as "more socially responsible" is a type of appeal that evidence suggests may fail to raise vaccination.

The same logic applies to smoking, they write. A chatbot could accurately say that most smokers never develop lung cancer, which may reassure a smoker and reinforce the habit. Another could accurately stress the risks and point to effective ways to quit.

Fishman and Jackson propose "behavioral benchmarking." Randomized experiments would test whether AI answers make people more or less likely to protect their health. They would start in areas of strong scientific consensus, such as smoking cessation and childhood vaccination, with results published so hospitals, insurers and consumers can compare models. This is a proposal, not a tested standard.

Next to watch is whether developers or independent groups publish randomized tests of how chatbot answers shape health intentions, alongside accuracy evaluations.

Sources

Source 1: https://pubmed.ncbi.nlm.nih.gov/42286322/

Source 2: https://pmc.ncbi.nlm.nih.gov/articles/PMC13375586/

Source 3: https://doi.org/10.1038/s41591-026-04431-5

Source 4: https://www.statnews.com/2026/11/10/ai-health-advice-accuracy-messaging-risk-behavioral-benchmarking/

Read this article online

27 / MARKETS & STRATEGY

Elysium Health Plans Six Additional Peptides Under New HHS Policy

Elysium Health, a longevity supplement company, plans to offer six additional peptides by prescription before the year ends, relying on a new federal enforcement policy.



Photo: “Omega 3 capsules in white bottle (52715127894)” by Jernej Furman from Slovenia, CC BY 2.0

The peptides are BPC-157, Epitalon, KPV, MOTS-c, Semax and TB-500. They will be offered through Elysium Health Rx, according to the company's November 5 announcement. Access will depend on applicable regulations and an individual evaluation by a board-certified physician.

The opening is a policy, not an approval. The U.S. Department of Health and Human Services issued an interim enforcement-discretion policy covering the six peptides under specified conditions. These include qualified manufacturing, testing, patient-specific prescriptions and adverse-event reporting. The policy does not constitute FDA approval. Compounded drugs are not reviewed by the FDA for safety, effectiveness or quality.

Elysium says the policy matches recommendations it made at a July advisory meeting. That claim is the company's own.

The company is pairing the retail move with two research efforts. The first is the Elysium Peptide Evidence and Evaluation Resource, or PEER. It is meant to give qualified research institutions access to rigorously characterized peptides through a controlled supply chain.

The second is TIME-PEP, a planned study through the company's Aging Research Center. It will look at epigenetic changes, measures of biological age, and possible associations with morbidity and mortality. Elysium says human clinical evidence for these peptides is limited. The announced research program does not establish that the products improve health or extend life.

Whether the six peptides reach Elysium Health Rx by year-end, what TIME-PEP enrolls and measures, and how other longevity companies respond to the HHS conditions.

Sources

Source 1: https://www.prnewswire.com/news-releases/elysium-health-expands-access-to-six-additional-peptides-under-new-hhs-policy-and-announces-launch-of-peptide-research-programs-302930925.html

Source 2: https://www.hhs.gov/sites/default/files/hhs-503a-peptide-enforcement-discretion-policy-appendix-b.pdf

Source 3: https://www.govinfo.gov/content/pkg/FR-2026-10-30/html/2026-22192.htm

Read this article online

28 / MARKETS & STRATEGY

OIG Finds Indiana Public Entities Recouped $205 Million Intended for Nursing Homes

Indiana nursing facilities did not retain $205.2 million in Medicaid supplemental payments intended to increase their reimbursement. Nonstate government-owned entities recovered the money instead, according to a November report from the HHS Office of Inspector General.

The report found that nonstate government-owned entities, such as county hospitals, recovered $479.8 million of Indiana's supplemental payments. That is nearly half of the state's total. Of that sum, $274.6 million was the state share that those entities had first contributed.

The structure behind the flows matters for operators. During the audit period, nonstate government-owned entities, including county hospitals, owned or operated about 97% of Indiana nursing facilities, according to OIG.

Scale explains why OIG looked at Indiana. In fiscal 2022 the state received about $1.02 billion of the $2.36 billion in nursing facility supplemental payments reported nationwide. That was the highest of any state. A 2020 Indianapolis Star investigation helped prompt the audit. The paper reported that officials across Indiana acquired the licenses of private nursing homes, which gave them access to extra Medicaid funding available only to government-owned nursing homes.

OIG also found errors in how the state handled the payments. State officials failed to report overpayments, carried an extra leap-year day into a non-leap-year calculation and used an incorrect federal matching rate. Indiana claimed $12.2 million in federal payments that did not comply with federal and state requirements.

OIG made four recommendations. They include exploring ways to increase the share of supplemental payments that participating nursing facilities keep, and refunding the $12.2 million in excess payments to the federal government.

The report frames the issue as a national one, citing longstanding concerns about complexity, scale and limited transparency in Medicaid supplemental payments. It points to a 2019 Senate Finance Committee report on the same subject.

OIG explicitly noted that nothing prohibited the entities from recovering the $205.2 million, although that money did not increase nursing facility reimbursement as intended. This finding is distinct from the $12.2 million in federal payments that did not comply with requirements.

Indiana did not indicate concurrence or nonconcurrence with the recommendations. It said it would consider ways to increase the share retained by nursing facilities and described steps taken or planned in response to the other recommendations.

Sources

Source 1: https://oig.hhs.gov/documents/audit/11972/A-05-24-00017.pdf

Read this article online

29 / MARKETS & STRATEGY

Ionis Cuts Tryngolza Sales Forecast by Roughly Half

Ionis reported $9 million in third-quarter U.S. net product sales for Tryngolza and lowered its 2026 forecast to approximately $50 million.

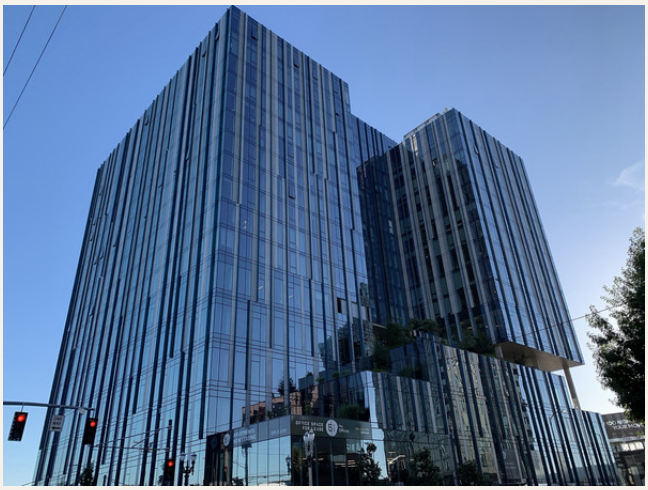


Photo: “5 MLK building Portland 2025” by PortlandAppraisalBlog, CC BY-SA 4.0

Ionis now expects Tryngolza, its triglyceride-lowering drug, to bring in approximately $50 million in 2026 U.S. net product sales, according to its November 9 financial release. The company's earlier forecast was $100 million to $110 million. That is a cut of roughly half.

Third-quarter U.S. net product sales were $9 million, bringing the first nine months of 2026 to $41 million. Ionis attributed the lower forecast to a more gradual launch ramp than anticipated.

For investors, the episode is a reminder of how much an early sales path can matter. Peak-sales estimates for new drugs rest on assumptions about how quickly clinicians adopt them.

Ionis said it remained confident in Tryngolza's long-term growth potential and multibillion-dollar peak sales. Those are company expectations, not achieved revenue.

Whether Ionis reports prescriptions rising in coming quarters, and whether its long-term sales expectations change.

Sources

Source 1: https://ir.ionis.com/news-releases/news-release-details/ionis-reports-third-quarter-2026-financial-results-and

Read this article online

30 / MARKETS & STRATEGY

Long-Term Unemployment Reaches 32% of Jobseekers Aged 55 and Older, AARP Data Show

Nearly one in three unemployed Americans aged 55 and older has been looking for work for at least 27 weeks, according to the AARP Public Policy Institute's digest of October 2026 Bureau of Labor Statistics data.



Photo: “Job Listings” by Flazingo Photos, CC BY-SA 2.0

The digest, published November 6, puts the long-term unemployed share of jobseekers 55 and older at 32 percent. For jobseekers aged 16 to 54, the share was 28.1 percent. The report defines long-term unemployment as 27 weeks or more of job searching.

The overall unemployment rate for workers 55 and older rose to 2.8 percent in October from 2.6 percent in September, according to AARP's report. About 1.1 million people in this age group were unemployed. The report counts 38.3 million workers 55 and older as employed.

The change was not even across sexes. According to AARP, the unemployment rate for women 55 and older rose from 2.2 percent to 2.9 percent. For men 55 and older, it fell from 3 percent to 2.8 percent.

Participation moved up as well. AARP reports that labor force participation for those 55 and older rose from 36.9 percent to 37.2 percent. The unadjusted rate for those 65 and older rose from 18.4 percent to 18.7 percent. More older people were working or actively looking for work.

The broader market was mixed, according to the digest. The economy added 165,000 jobs in October, up from 60,000 in September, which was revised up from 29,000. The overall unemployment rate edged down from 4.2 percent to 4.1 percent. Job gains came in professional and business services, health care, transportation and warehousing, and manufacturing. BLS described construction, wholesale trade, and leisure and hospitality employment as showing little change over the month; small point estimates should not be treated as clear sector trends.

For employers, the figures point to a pool of experienced candidates who are searching longer than younger peers. The digest notes that jobseekers 55 and older typically have higher long-term unemployment than younger groups.

The figures come from the Bureau of Labor Statistics' monthly Employment Situation release, which draws on two surveys: the payroll survey and the household survey. Next month's release will show whether the rise in older women's unemployment and the 32 percent long-term share persist.

Sources

Source 1: https://www.aarp.org/pri/topics/work-finances-retirement/employers-workforce/employment-data-digest/

Source 2: https://www.bls.gov/news.release/archives/empsit_11062026.htm

Read this article online

31 / LONGEVITY & HEALTHSPAN · LONG READS

Fat Molecules Get Longer With Age Across Species, and Reversing It Extended Worm Lifespan

A Nature Aging study finds lipid chains lengthen with age in mice, worms, flies and people. Blocking one enzyme reversed this in worms and helped them live longer. Human benefit is untested.

Every cell membrane is built from fats, and those fats come in different lengths. A new study finds that, with age, the balance shifts toward longer chains. The pattern shows up in mice, worms, flies and humans. In worms, pushing it back extended life.

Our research desk reported that researchers in Nature Aging found lipid chains grow longer with age across four species. Longer chains also appeared as human heart disease progressed, and dietary restriction shortened heart lipids in mice.

The desk said an enzyme called Plb1 regulates this remodeling. Its expression correlated with lifespan in mice, and genetic analyses supported a causal role in human frailty. Reducing Plb1 in worms reversed the lengthening and extended lifespan.

According to PubMed, the study, "Longer lipids mark aging and constrain lifespan," was published October 6 in Nature Aging. The senior author is Georges Janssens of Amsterdam UMC. Co-authors include aging researchers Linda Partridge and Johan Auwerx.

The team measured lipids across many tissues and species. With age, longer lipid chains rose and shorter ones fell. The authors call it a ratio shift, not just an addition of long chains.

The author list spans labs in Amsterdam, Lausanne, Cologne, Sheffield, Luxembourg, Maastricht, Düsseldorf, Ohio and California. That breadth matters. Different groups supplied different species and tissues, which makes the shared pattern harder to dismiss as one lab's quirk.

The heart findings came from human tissue. Lipid chains lengthened as heart disease progressed. In mice, eating less shortened heart lipids, which fits with dietary restriction's known effects on aging in animals.

The causal step was in worms. Knocking down Plb1 reversed lipid lengthening and extended lifespan. The benefit depended on lipid length, which supports a role for chain length in that experimental system. It does not establish a human treatment.

This adds a candidate hallmark of aging: a measurable, shared shift in membrane fats. Because it appears across species and in diseased human hearts, it could become a marker worth tracking.

Nobody has shown that shortening lipids improves health in people.

Worm results also translate poorly. Many interventions that extend worm life do little in mammals. The next real test is whether lowering Plb1 helps mice live longer or healthier.

• Do not buy supplements marketed as "lipid shortening" or Plb1-targeting. No human evidence supports them. • Keep to proven basics for heart and metabolic health: regular exercise, sleep, and a diet that avoids chronic overeating.

• If you are considering calorie restriction or long fasting, talk with your doctor first, especially if you are older, underweight, or have diabetes or heart disease.

• Open question: whether reducing Plb1 helps mammals, and whether doing so carries harms.

• Mouse studies that lower Plb1 and measure lifespan and healthspan. • Whether blood lipid chain length can serve as an aging marker in human cohorts. • Independent replication of the cross-species lipid pattern.

Sources

Source 1: https://pubmed.ncbi.nlm.nih.gov/42839094/

Source 2: https://doi.org/10.1038/s43587-026-01223-x

Source 3: https://eprints.whiterose.ac.uk/id/eprint/246495/

Read this article online

32 / LONGEVITY & HEALTHSPAN · LONG READS

Two Brain-Aging Studies Are Listed: Baltimore PET Imaging and a VR Driving Game

One study plans to follow brain amyloid and tau in 400 healthy older volunteers for years; the other tests whether a virtual driving game helps 20 people with advanced dementia. Neither has results yet, and they answer very different questions.

The amyloid plaques of Alzheimer's disease start building years before memory slips. Two registered studies attack cognitive aging from opposite ends. One watches the brain change in people who are still well. The other tries to bring a moment of engagement to people who are already deeply affected.

Our research desk flagged two entries on ClinicalTrials.gov. The first is a PET imaging study run inside the Baltimore Longitudinal Study of Aging, sponsored by the National Institute on Aging. The second is a University of Manitoba study of an augmented virtual reality driving simulator for people living with dementia in care.

Neither registry record has posted results. Registry absence alone does not establish that no findings have been presented elsewhere.

THE FEDERAL BRAIN-IMAGING COHORT

The registry record, NCT07099053, lists the National Institute on Aging as sponsor and a target of 400 participants. The record has been retitled "PET Imaging in the Baltimore Longitudinal Study of Aging (BLSA)" and now includes tau PET with [18F]florquinitau (Tauklarify) alongside florbetaben amyloid PET, so it is no longer an amyloid-only study. Volunteers must be at least 55 and already enrolled in the Baltimore study, which has followed healthy adults since 1958.

Participants get PET/CT scans with intravenous tracers that make amyloid and tau visible. Scans recur every one to four years depending on age, and yearly from age 80. Each scan takes roughly 30 minutes of lying still. The November 10 registry update lists the study as recruiting and gives November 15, 2026 as its start date. Recruitment status and the first imaging date are different facts. Estimated completion is December 12, 2029.

The value here is the setting, not the scan. The Baltimore study already collects detailed cognitive, physical and medical data on the same people over decades. Adding repeated PET imaging lets researchers ask when amyloid appears, how fast it builds, and whether it tracks with brain structure, cognition or other illness.

THE VR DRIVING STUDY

The Manitoba record, NCT06419257, plans to enroll 20 residents of a dementia care unit at Riverview Health Centre in Winnipeg. Participants must be 50 or older, mobile and able to see. Wheelchair users are excluded.

The simulator is built into an existing car body, and the study will likely use only its easiest level. The outcomes are soft. The narrative describes free-form nurse observations; the registered primary outcome uses a custom questionnaire scored worse, unchanged or improved, weekly for up to 52 weeks. Researchers infer implicit memory from in-game driving scores such as curb strikes and red lights run. The study began in January 2024, and the registry lists study completion in June 2027.

Amyloid now matters clinically in a way it did not five years ago. Anti-amyloid antibodies are approved for early Alzheimer's disease, and blood tests for amyloid are entering practice. Knowing how amyloid behaves in ordinary aging adults is what lets doctors interpret a positive result in a healthy 70-year-old.

Long cohorts like Baltimore's are where those reference curves come from. They are slow, unglamorous and enormously useful.

The VR study speaks to a different need: quality of life for people whose disease is advanced. A familiar activity like driving may prompt engagement or calm. That is worth testing, but it is not a treatment for dementia.

• Success in a game is not success in life. Better driving scores could simply reflect practice on that game. Our desk made the same point, and it is the right one. • Twenty participants with no control group listed, and mood assessed through nurse observations and a custom questionnaire, can generate ideas but not proof. • The Baltimore imaging study is observational. It can show associations between brain changes and decline, not cause and effect. • The Manitoba record lists primary completion in May 2027 and study completion in June 2027. Both dates are correct; they mark different milestones.

• If you are curious about your own amyloid status, ask your physician whether testing would change any decision for you. Without symptoms, it often does not. • Keep doing the proven basics for brain health: blood pressure control, physical activity, hearing correction, sleep and social contact. • If a loved one is in memory care, ask staff about structured, familiar activities. Judge them by mood and engagement, not by game scores. • Anyone with new memory or thinking changes should see their doctor for a full evaluation.

• Early Baltimore amyloid and tau analyses, likely years away, showing brain trajectories in healthy older adults.

• Whether the Manitoba team reports outcomes beyond the game itself, such as agitation or caregiver burden. • How amyloid blood tests are calibrated against PET in cohorts like this one.

Sources

Source 1: https://clinicaltrials.gov/study/NCT07099053

Source 2: https://clinicaltrials.gov/study/NCT06419257

Read this article online

33 / LONGEVITY & HEALTHSPAN · LONG READS

Evipedia Puts 860-Plus AI-Written Longevity Evidence Reviews on Hugging Face, Free to Download

The Forever Healthy Foundation now publishes its whole review library as an open dataset, refreshed daily. That is useful for builders and curious readers, but open access is not the same as proven accuracy.

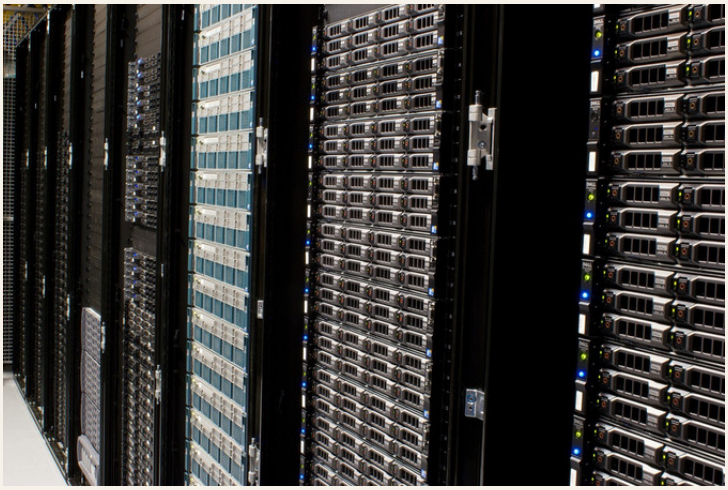


Photo: “Wikimedia Foundation Servers-8055 08” by Victorgrigas, CC BY-SA 3.0

Most longevity advice online is a claim without a paper trail. A nonprofit is now trying the opposite: hundreds of structured evidence reviews, each with an audit log, released as an open dataset anyone can download. The question is whether the reviews are good, not just whether they are free.

Our research desk reported that Evipedia, a library of longevity evidence reviews, had come to Hugging Face, the main public hub for AI models and datasets. It said the library holds more than 860 reviews of health and longevity interventions.

The desk noted what it did not know: access terms, update frequency and how the reviews judge study quality. It also listed weekly price moves for several gene-editing and longevity stocks.

The announcement is confirmed; its review count describes the October release, not an independently counted November total. A press release from the Forever Healthy Foundation, posted on Lifespan.io on October 7, 2026, says the full Evipedia dataset is now on Hugging Face. The foundation describes itself as a private humanitarian initiative focused on extending healthy lifespan.

The release fills in the gaps our desk flagged:

• Size: more than 860 reviews in the summary, 870-plus in the feature list. • Format: one row per review, with metadata and the full text in Markdown, aimed at machine learning and retrieval pipelines. • Updates: the dataset refreshes daily; individual reviews are revisited about

every four to six weeks. • Method: reviews are generated with frontier AI models using an open-source framework called AI4L and a process the foundation calls Audit-Driven Prompting. • Transparency: each intervention gets a one-page quick reference and a full review, each with an audit report listing criteria and the history of fixes. The dataset card explicitly licenses the catalogue under CC BY 4.0, with attribution required.

Readers of this newsletter ask about rapamycin, NAD boosters, metformin, saunas and dozens of other interventions. A searchable, consistent library of evidence summaries is a real convenience. It also lets developers build tools that cite a reviewed source instead of a random web page.

The audit trail is the most promising part. A review that shows its criteria and its corrections is easier to trust, and easier to challenge, than one that does not.

The reviews are written by AI. The foundation calls them hallucination-free. That is a strong claim, and we found no independent validation of it. An audit process that also runs on AI may share the blind spots of the model it checks.

A large library is not proof of a reliable one. What matters is how each review grades study design: whether it separates mouse data from human trials, and observational links from randomized evidence.

As for the stock moves our desk listed alongside the story, we did not verify them. They came from an internal price feed, and nothing ties them to this announcement. We leave them out.

• Use Evipedia as a starting map, then open the underlying studies for anything you plan to act on. • Check whether a review's key evidence comes from humans, animals or cells before weighing it. • Bring a review to your physician rather than starting a supplement or drug on its strength alone, especially if you take prescription medicines.

• Independent spot-checks of Evipedia reviews against expert systematic reviews. • Compliance with the dataset’s CC BY 4.0 attribution requirements when reusing it. • Whether clinicians or medical societies begin citing it, or flag errors in it.

Sources

Source 1: https://lifespan.io/evipedia-goes-hugging-face/

Source 2: https://huggingface.co/datasets/forever-healthy/evipedia-reviews/blob/main/README.md?code=true

Read this article online

34 / MARKETS & STRATEGY · LONG READS

Deno's Team Joins Cloudflare, and Profitable Oxide Raises $445 Million

The JavaScript runtime company is folding into Cloudflare, winding down its hosting service. Server maker Oxide raised a Series D because orders outrun supply. Cloudflare's post carries an Acquisitions tag, though neither company's text uses the word.

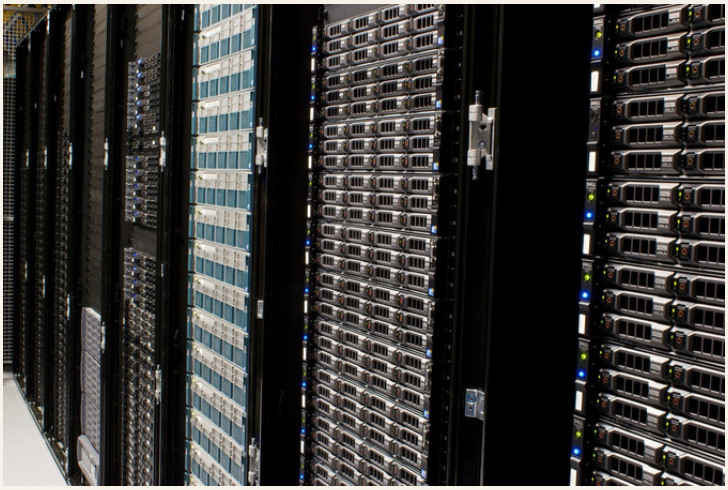


Photo: “Wikimedia Foundation Servers-8055 08” by Victorgrigas, CC BY-SA 3.0

Two announcements on October 9 arrived together. One company's team joined a giant; the other raised a large round because it could not build machines fast enough.

Cloudflare and Deno published matching posts on October 9, 2026. Deno creator Ryan Dahl wrote that the entire Deno team is joining Cloudflare. Neither side disclosed financial terms, and neither post uses the word acquisition in its body text. The Cloudflare post is filed under an 'Acquisitions' tag, and The New Stack, BetaNews and others reported the deal as an acquisition.

The goal is to make Cloudflare's Workers programming model a mainstream way to build servers. Dahl and Bert Belder will lead an effort to make self-hosting workerd, Cloudflare's open-source runtime, a supported option. They will merge in ideas from celld, a self-hostable system Deno released in August.

There are real consequences for current users. According to Deno's post, Deno will continue monthly bug-fix and security releases of the runtime for one year, after which development ends. The runtime stays open source. Deno Deploy, the company's hosting service, will run for six months and then shut down, with migration help for paying customers. The JSR package registry continues on Cloudflare infrastructure.

celld matters to the logic of the deal. Deno creator Ryan Dahl describes it as a single Rust program whose only outside dependency is object storage. It lets developers run Workers-style distributed applications on their own machines. Folding it into workerd gives Cloudflare a credible answer for customers who want the same programming model off its cloud.

Oxide Computer, which builds rack-scale servers for companies that run their own data centers, announced a $445 million Series D on October 9. Eclipse led. USIT, Riot Ventures and Jane Street took large allocations. Atreides Management joined as a new investor, and AMD came in as a strategic investor.

The reason for raising is unusual. Oxide says it paid income tax in the spring, earned from ordinary sales after component, manufacturing and salary costs. It describes a very large order backlog, with demand far exceeding supply. Hardware requires paying for parts and manufacturing long before customers pay. Without new capital, Oxide says, it would have had to be cautious about taking new orders.

The pace is notable. Oxide announced a $200 million Series C in February 2026, so this round arrives about eight months later. In a company FAQ, CEO Steve Tuck said Oxide turned profitable and that demand continues to outstrip supply. The constraint, he suggested, is securing parts such as memory, storage and electrical components.

Medical AI, hospital software and research computing all sit on this layer. When a platform absorbs an independent runtime, users gain stability but lose a choice, and the Deno runtime's development ends after one year.

Oxide's claims about profit and backlog are company statements; it gave no figures. Treat them as signals, not audited facts. Not financial advice.

• Our summary said Cloudflare acquired Deno. The companies describe the team joining Cloudflare and disclose no deal terms. • The summary omitted that Deno Deploy will shut down in six months and runtime development will end after a year of maintenance releases. • The summary said Oxide would have had to ration orders; Oxide's wording is that it would need to be cautious about accepting new demand.

• How Deno Deploy customers migrate before the six-month shutdown. • Whether a community fork keeps the Deno runtime developing after next year. • Oxide's manufacturing ramp, and whether large health systems appear among its customers.

Sources

Source 1: https://deno.com/blog/cloudflare

Source 2: https://blog.cloudflare.com/deno-joins-cloudflare/

Source 3: https://oxide.computer/blog/our-445m-series-d

Source 4: https://oxide.computer/faq-friday/why-did-oxide-raise-445m

Read this article online

35 / MARKETS & STRATEGY · LONG READS

Big Pharma Filled the Speaker List at a Major Aging-Drug Meeting

The 2026 Aging Research and Drug Discovery meeting at Harvard drew speakers from more than a dozen major drug companies, the FDA and big investors. Interest in aging biology is real; an approved aging drug is still years away. The organizer describes the meeting as the world's largest dedicated to longevity biotechnology, a framing no independent count backs up.



Photo: “Boulder field near Cap Rock under storm clouds (52352558862)” by Joshua Tree National Park, Public domain

A decade ago, aging research meetings were smaller affairs. In October, the speaker list at a major aging-drug meeting read like a pharma industry directory. That signals attention, not proof that investment or clinical outcomes will follow.

The 13th Aging Research and Drug Discovery meeting, known as ARDD, ran October 1 to 3, 2026, at Harvard's David Rubenstein Treehouse in Boston. It is organized by Insilico Medicine, an AI drug discovery company. Its chairs include Vadim Gladyshev of Harvard Medical School and Alex Zhavoronkov of Insilico.

Eli Lilly and Insilico were the top-tier sponsors. AbbVie, BioAge, Human Longevity and others sponsored at lower tiers. McKinsey Health Institute was the knowledge partner.

The published speaker roster includes scientists and executives from:

The published program includes speakers from Lilly, Novartis, Pfizer, Takeda, GSK, AstraZeneca, Novo Nordisk, Genentech, Roche, Astellas, Daiichi Sankyo and Lundbeck. The October 2 Pharma Chief Executive Panel, moderated by TIME's Alice Park, lists Fiona Marshall, Ariel Feldstein, Elcin Barker Ergun, Christophe Weber and Stéphane Bancel. Other sessions include FDA officials and ARPA-H, alongside academic researchers and biotechnology companies.

Speaking slots do not equal strategy, but deals do. In March 2026, Insilico announced a research collaboration with Lilly worth up to about $2.75 billion, including $115 million upfront, according to the company's release.

Some outlets framed that deal as Lilly's big bet on aging. The release itself never uses the word. It names multiple therapeutic areas. That gap is typical: pharma studies aging biology but files for specific diseases.

Large pharmaceutical companies have the resources to run long, costly outcome trials. Their presence creates an opportunity for better evidence, but it does not guarantee trials, approvals or insurance coverage.

The heavy FDA presence is also notable. Regulators showing up to an aging meeting suggests the conversation about biomarkers and new endpoints is active, even if rules have not changed.

The program itself pointed toward the clinic. The first day held a longevity medicine forum and a workshop for the XPRIZE Healthspan competition, alongside several clinical-trial and regulatory sessions. Later days covered AI in drug discovery, virtual models of aging cells and even pet longevity, a field where some aging drugs may reach the market first.

For patients, the practical upshot is modest for now. A conference program does not establish that an intervention works or predict when it might reach a pharmacy.

• Sponsorship shapes the stage. The organizer and top sponsor are business partners. • Open question: whether trials of drugs that affect aging biology will demonstrate meaningful clinical benefits beyond their established indications.

• Follow-up research and trial reports from ARDD 2026 speakers. • Any FDA statement on biomarkers or endpoints for aging trials. • First clinical candidates from the Lilly–Insilico collaboration. • Whether more pharma companies form dedicated aging units. Not financial advice.

Sources

Source 1: https://agingpharma.org/program2026

Source 2: https://www.agingpharma.org/speakers2026

Source 3: https://insilico.com/news/tyem531fm1-insilico-medicine-announces-ardd-2026-pr

Source 4: https://insilico.com/news/uiy12zcjg1-insilico-medicine-announces-global-rampd

Read this article online

36 / MEASUREMENT & AI

Google Launches Gemini 4 Argon; AI Weather Models Add a Day of Warning

Several of the Google releases we tracked were weeks older than our coverage suggested. The weather work is the part with peer-reviewed evidence and a direct link to health.



Photo: “Boulder field near Cap Rock under storm clouds (52352558862)” by Joshua Tree National Park, Public domain
Photo: “NOIRLab HQ Server Racks (6V6A0375-CC)” by NOIRLab/NSF/AURA/T. Slovinský, CC BY 4.0

Between late September and early October, Google appeared in our coverage five times: a new family of Gemini 3.8 models, a weather model that gives an extra day of cyclone warning, and finally a new flagship, Gemini 4 Argon. The underlying news is mostly real. The timeline is not, and sorting it out shows what actually changed.

On Sept 24 and 26 our research desk reported a single-day launch of Gemini 3.8: a fast Flash model, a security-focused Flash Cyber, a Live voice mode with a moving avatar, a new text-to-speech voice and a small on-device model. On Sept 26 it reported a Nature study showing Google's WeatherNext model adds at least a day of lead time on cyclones. On Sept 30 it noted a WeatherNext 3 release. On Oct 8 it reported the launch of Gemini 4 Argon.

GEMINI 3.8 WAS AN EARLY-SEPTEMBER RELEASE

Gemini 3.8 Flash and 3.8 Flash Cyber launched on Sept 2, according to Google’s September 2 announcement. Flash Cyber is restricted to vetted security teams in Google's Fairwind Program. So the wave our desk called new on Sept 24 was about three weeks old. We could not independently confirm the release dates or details of the Live avatar mode and the Omni 1.1 Flash model.

THE CYCLONE RESULT IS SOLID, AND OLDER

The peer-reviewed cyclone result was announced on August 6. The paper's abstract reports an average lead-time advantage of one day or more over leading operational models in evaluations of 2023–2025 cyclones. Google's accompanying release describes three-day forecasts matching prior systems' two-day accuracy, with approximate three-day errors of 100 kilometers for track and 11 knots for intensity. This review checked the paper's abstract and the Google-authored release, not the full paper.

It is also in real use. Google says the model helped the US National Hurricane Center anticipate Hurricane Melissa's rapid strengthening before landfall in Jamaica in 2025. Google released the code and weights openly.

The Sept 26 item linked that Nature study to WeatherNext 3. They are different models. WeatherNext 3 was announced on Sept 3, according to Google, and Google said it would begin powering Search, Maps and the Gemini app from that day. Its headline claim, up to 60 percent improvement in a probabilistic rainfall score against the IMERG dataset, comes from Google's own evaluation, not independent testing.

GEMINI 4 ARGON IS REAL AND LAUNCHED SEPT 30

Our desk reported Argon on Oct 8, but Google dates the announcement to Sept 30, with initial access limited to Fairwind Program cyber partners. Google made Argon generally available on Oct 13: the API in AI Studio and the Gemini platform, Ultra in Antigravity, and Pro and Ultra in the Gemini app and AI Mode in Search.

Google lists introductory API pricing of $2 per million input tokens and $10 per million output tokens, rising to $4 and $20 after November 30, 2026. Its September 30 announcement reports a 77.9 percent score on DeepSWE v1.1. That is a company benchmark result, not evidence of clinical safety.

The chatbot race gets the headlines, but the weather models matter more to health. Heat waves, floods and hurricanes drive emergency visits, dialysis and oxygen disruptions, and deaths among older adults. An extra day of reliable warning gives families time to evacuate, refill medications and move vulnerable relatives. Hospitals get time to protect power and staffing.

The Argon story matters differently. New frontier models change the cost and capabilities of tools built on top of them. Neither software benchmarks nor contractual health-data protections establish that a clinical use is safe.

• If you or a parent live in hurricane or heat country, sign up for local emergency alerts and keep a written medication list and a week's supply ready. • People on home oxygen, dialysis or insulin should have a power-outage plan agreed with their care team before storm season. • Treat new model benchmarks as marketing until independent groups repeat them.

Will WeatherNext 3's rainfall gains hold up in rare, dangerous storms when tested independently? Will Argon's coding and security results survive outside testing? And how will Google explain weather-forecast uncertainty to the public once it appears in Maps and Search?

• Independent scoring of WeatherNext 3 on benchmark suites such as WeatherBench. • National Hurricane Center use of the cyclone model through the rest of the 2026 season. • Third-party

benchmark results for Argon following its Oct 13 general release, and what happens to usage when pricing rises after Nov 30. • How WeatherNext forecasts show uncertainty as they appear in Maps and Search.

For official forecasts, severe-weather warnings and public safety advice, use your local meteorological agency or national weather service.

Sources

Source 1: https://blog.google/innovation-and-ai/models-and-research/gemini-models/3-8-flash-and-3-8-flash-cyber/

Source 2: https://blog.google/innovation-and-ai/models-and-research/gemini-models/gemini-4-argon/

Source 3: https://blog.google/innovation-and-ai/models-and-research/gemini-models/introducing-gemini-4-argon/

Source 4: https://blog.google/innovation-and-ai/models-and-research/google-deepmind/introducing-weathernext-3/

Source 5: https://pubmed.ncbi.nlm.nih.gov/42717078/

Source 6: https://www.preventionweb.net/news/weathernext-ai-model-achieves-breakthrough-forecasting-cyclones

Read this article online

37 / MEASUREMENT & AI

Frontier AI Prices Fell Sharply in Weeks, Changing What Medical AI Can Afford

Official price lists show cheaper input and output tokens on several models. Workload savings, longer prompts and subscription availability require a closer look.

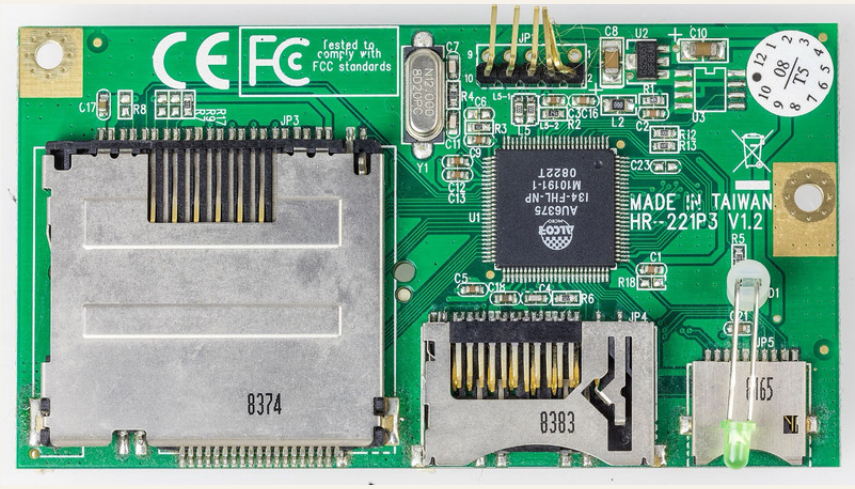


Photo: “5.25- computer bay interface unit - board 1-8011” by Raimond Spekking, CC BY-SA 4.0 Photo: by James St. John, CC BY 2.0

Several September and October releases lowered AI token prices. A small model from a frontier family now lists at a dime per million input tokens. But token prices alone do not establish the cost, reliability or clinical value of a finished task.

Our research desk tracked the price story in September and October. Official price lists support the broad direction, but some headline savings mix list prices, workload estimates and temporary promotions.

ANTHROPIC'S OPUS 5.5, RELEASED SEPT 22

Opus 5.5 lists at $4 per million input tokens and $20 per million output, with cached reads at 20 cents, according to Anthropic. That is 20 percent below Opus 5's list price of $5 and $25. Cache reads are 60 percent lower.

The 40 percent figure is real but narrower than a list-price reduction: it is Anthropic's estimate for typical workloads at default settings. Tasks can use different numbers of tokens and cost more or less than that average. Sonnet 5.5 currently lists at $2 per million input tokens and $10 per million output tokens.

OPENAI’S CURRENT SOL AND LUNA PRICES

OpenAI's current standard API price for GPT-6.1 Sol is $2 per million input tokens and $10 per million output tokens. Cached input is $0.10, a 95 percent discount from standard input. GPT-6 Luna lists at $0.10 input, $0.50 output and $0.01 cached input per million tokens. Faster service tiers and some regional-processing options cost more.

ANTHROPIC'S HAIKU 5.5, RELEASED OCT 7

Haiku 5.5 costs 10 cents per million input tokens and 50 cents per million output for prompts up to 100,000 tokens, according to Anthropic. Longer prompts cost five times more. The previous Haiku 4.5 cost $1 and $5, so short-prompt prices fell by 90 percent. Anthropic says the cheaper tier covers about 90 percent of past Haiku requests.

CHATGPT AVAILABILITY IS A SEPARATE QUESTION

API pricing does not establish what model an individual gets in ChatGPT. The current OpenAI Help page says GPT-6 access depends on plan and rollout, while its Free and Go guidance still names GPT-5.6 Luna. We cannot confirm a universal GPT-6 default for free accounts from that page.

The September items were about flagship prices. By October the story had moved down the range, to small, fast models with much lower token prices. Cheap tokens do not remove the need to test accuracy, permissions and privacy.

Many proposed medical AI uses involve long documents. Summarizing a decade of records, checking a medication list against guidelines, or monitoring wearable data around the clock means processing many tokens, often repeatedly. Cache discounts matter most here, because a patient's history is reread at each step.

At these prices, some tasks may become more affordable for clinics, insurers and individuals. That can widen access. It also means much more AI-generated health advice, of uneven quality, reaching people who have no clinician reviewing it.

• Use free AI to prepare for appointments, such as listing questions or summarizing your own records, not to replace them. • Be wary of health apps that do not name their underlying model or say how outputs are checked. • Never change a medication based on chatbot output without your physician.

The 40 percent saving on Opus 5.5 is a workload estimate, not a price-list fact. Anthropic puts Haiku 5.5's average running-cost reduction at about 75 percent, not the 90 percent short-prompt token-price cut. Its documentation also warns that newer tokenizers can produce about 30 percent more tokens for the same text, depending on content.

• Whether GPT-5.6 Sol promotional pricing changes after November 30, the end date on OpenAI's current pricing page. • Independent cost-per-task and accuracy tests, using the same workloads and settings. • How health products using these models validate outputs and protect patient information.

Sources

Source 1: https://www.anthropic.com/claude-opus-5-5

Source 2: https://www.anthropic.com/claude-haiku-5-5

Source 3: https://platform.claude.com/docs/en/about-claude/pricing

Source 4: https://openai.com/index/introducing-gpt-6-1-sol/

Source 5: https://developers.openai.com/api/docs/pricing

Source 6: https://help.openai.com/en/articles/20001354-gpt-6-and-other-models-in-chatgpt

Read this article online

38 / SCIENCE & FRONTIERS

Uranium Slowly Loses Its Easy Mode, and the Physics Says the Clock Is Real

Our research desk proposes that uranium decay could make unenriched reactors harder for a much later civilization to build. The isotope arithmetic is straightforward; the reactor-design and historical consequences remain hypotheses.



Photo: by James St. John, CC BY 2.0 Photo: “Разнообразие почвенной микрофлоры в чашке Петри” by Oksana Lastochkina, CC BY-SA 4.0

Earth’s primordial uranium predates the planet, and the most useful kind is quietly disappearing. About two billion years ago, uranium ore in present-day Gabon ran as a natural nuclear reactor with nothing but groundwater to help. Today the isotope mix is much less favorable for an Oklo-like reactor, and in another two billion years it may be impossible to build one on purpose without enrichment.

That is the core of a proposal from our research desk. It asks whether the age of a planet, not just its resources, decides which technologies a society can reach first.

Uranium comes in two main forms. Uranium-235 sustains a chain reaction readily and has a half-life of about 704 million years. Uranium-238 is far more stable, with a half-life near 4.47 billion years. So the share of the useful isotope falls steadily with time.

Natural uranium today holds about 0.72 percent uranium-235. That is just enough for heavy-water and graphite reactors to run on unenriched fuel. Ordinary light-water reactors need enrichment.

The desk's prediction: a careful reactor-physics comparison will show that uranium aged another two billion years, at roughly 0.14 percent uranium-235, cannot sustain a chain reaction in any of those thermal designs without enrichment or outside help. This is an untested reactor-design hypothesis, not a calibrated probability. The broader idea that losing this option could delay a civilization is also uncertain.

Using a two-isotope decay model, a present uranium-235 fraction of 0.72 percent and half-lives of 704 million and 4.47 billion years gives about 0.138 percent after two billion years and 3.67 percent two billion years ago. These are calculated isotope fractions, not reactor-criticality results.

Oklo itself is well documented. A 2018 study in PNAS by Groopman and colleagues at the US Naval Research Laboratory found Oklo uranium with a uranium-235/uranium-238 ratio of 0.3655 ± 0.0007 percent (2σ), the most depleted natural uranium known. That deficit is the fingerprint of fission that ran long ago.

Uranium is not only a power source. Much of modern nuclear medicine depends on reactors. Technetium-99m, used in millions of heart, bone and cancer scans, comes from molybdenum-99, traditionally made by fissioning uranium targets in research reactors.

A 2012 review by Pillai and Knapp described recurring shortages of that supply and alternative routes that avoid uranium fission. The lesson is practical: diagnostic medicine rests on a small number of physical facilities and isotopes, and supply resilience is a real health issue.

There is also a lesson about judging claims. Isotope decay can be calculated from measured half-lives. Whether a particular fuel and reactor configuration sustains criticality requires a defined model, and any effect on civilization requires a separate historical argument.

The isotope trend is established; the proposed reactor limit and civilizational consequences have not been demonstrated here. A society that discovers fission could also learn isotope separation, or turn to thorium, or never need nuclear power at all.

Enrichment is hard, but humans solved it within a few years of discovering fission. So a late-arriving society would lose an easier starting route, not the technology itself. The proposal cannot measure how much that delay would matter.

• A published, reproducible reactor-physics comparison of today's uranium against the aged mixture. • New Oklo isotope work refining how long and how hot the natural reactors ran. • Progress on non-fission routes to molybdenum-99 that make medical imaging less dependent on uranium reactors.

Sources

Source 1: https://www.epa.gov/radiation/radionuclide-basics-uranium

Source 2: https://pubmed.ncbi.nlm.nih.gov/30104355/

Source 3: https://pubmed.ncbi.nlm.nih.gov/23013668/

Read this article online

39 / SCIENCE & FRONTIERS

Pioneer Labs Reports Bioplastic Production From Simulated Mars Resources

A September preprint reports more than three times the parent strain’s bioplastic titer in an evolved bacterium under simulated Mars conditions on Earth. It does not show life or manufacturing on Mars.



Photo: “Разнообразие почвенной микрофлоры в чашке Петри” by Oksana Lastochkina, CC BY-SA 4.0

A small nonprofit in Emeryville, California, says it has built the first microbe designed for Mars. Fed simulated Martian soil and air, it makes biodegradable plastic that could be printed into shelters. The work remains a preprint, and the company’s projections extend well beyond the laboratory results.

Our research desk reported that Pioneer Labs announced "this week" an engineered organism, sPL.001, that lives on chemically accurate Martian dirt, water and air, with toxins left in and no fertilizer, while making bioplastics. The announcement came from chief executive Erika DeBenedictis.

The company describes a heated bioreactor and a nonphotosynthetic organism supplied with acetate, a carbon source. Acetate does not supply nitrogen.

The company’s announcement is dated September 22, 2026, not this week.

sPL.001 is an evolved strain of Cupriavidus necator H16, a bacterium long known for making polyhydroxyalkanoates, a family of biodegradable plastics. The team used lab evolution over more than a trillion cell divisions to toughen it against acetate and leached Martian-like soil.

A bioRxiv preprint, "Bioplastic Production from simulated 100% in situ Mars resources" (doi 10.64898/2026.09.14.748363), screened 16 organisms, also evolved Pseudomonas putida KT2440 alongside C. necator, and reports more than three times the bioplastic titer of the parent strain under simulated Martian conditions on Earth. The microbe feeds on acetate made from

carbon dioxide. Our desk's phrase "nitrogen delivered as acetate" is chemically muddled: acetate supplies carbon, not nitrogen.

The company reports that the cells stop growing without supplied acetate and are killed by simulated Mars stressors. Its proposed “Propagation Restricted, Inert on Mars” framework is not an independently established regulatory approval or a demonstration of zero contamination risk on Mars.

Mars is far from most readers' plans. The tools are not. Directed evolution and containment strategies are the same methods used to make engineered microbes for medicine, from live bacterial therapies to drug manufacturing.

Showing that simulated soil provides nutrients is different from showing that an organism can grow in real Martian conditions. The containment claims also need independent scrutiny.

• "This week" is wrong. The announcement was September 22–23. • Earth-only results. All tests were simulations on Earth; Pioneer calls it a proof of concept, not flight-ready. • City in one launch. DeBenedictis says the equipment fits one rocket and could make materials for a small city. That is a projection, not a result. • Planetary protection. The only figure on offer is the company's own claim that a leak would clear thresholds "by a factor of several hundred."

• Peer review of the bioRxiv preprint and the companion biosafety paper. The inspected source is a preprint; an exhaustive later-publication and correction search was not completed. • Whether any space agency or launch company commits to flying the system.

Sources

Source 1: https://blog.pioneer-labs.org/p/announcing-the-first-microbe-for

Source 2: https://blog.pioneer-labs.org/p/when-is-a-microbe-safe-to-send-to

Source 3: https://doi.org/10.64898/2026.09.14.748363

Source 4: https://sciety.org/articles/activity/10.64898/2026.09.14.748363

Read this article online

40 / HISTORY & IDEAS

Did Storable Grain Make Conquered Land More Valuable to Early States? A Clear Claim With a Clear Test

Our research desk proposes that early rulers gained more from annexing grain land than from equally fertile land whose surplus spoiled quickly. This is a testable hypothesis, not an established conquest effect.

Two valleys can feed the same number of people and still be worth very different amounts to a ruler. One grows grain that keeps for a year in a granary. The other grows food that rots within days. Our research desk argues that this difference, not raw fertility, shaped which conquests made early states stronger.

It is a small, precise claim about the deep past. It also carries a lesson that applies well beyond archaeology: what an economy produces is not the same as what anyone can collect.

A government needs resources on a schedule. Soldiers and officials must eat between harvests. Builders need provisions while away from their own fields. Food that cannot be stored, measured and moved is hard to turn into a standing administration.

Dry grain solves those problems. It can be stored, divided into standard units and handed out over months. A tax collector can inspect a threshing floor or granary far more easily than he can estimate everything a household grows and eats.

So, the essay argues, annexing grain land did two things at once. It added food, and it made that food easier to collect regularly. That should have let a state support more permanent officials and soldiers than annexing land of equal output whose surplus spoiled.

The author is careful about limits. This is one advantage of conquest, not a claim that grain caused states. And storage techniques such as drying and fermenting can make 'perishable' crops keep, so any comparison must be about food as people actually handled it.

The core distinction is a familiar one in economics. Modern governments see it whenever an economy grows but tax receipts do not follow. Output and collectable revenue are different numbers.

The essay leans on two prior works. One is a 2022 economics paper by Joram Mayshar, Omer Moav and Luigi Pascali arguing that the ability to appropriate crops explains state formation better than farm productivity alone. The other is a 2017 book by the political scientist James C. Scott on grain and early states. The paper’s primary abstract and publication record are confirmed. It appeared online March 8, 2022 and in the April issue of the Journal of Political Economy. Across several datasets, the authors report a causal effect of cereal cultivation on hierarchy and no similar effect for land productivity.

Their mechanism is the one the essay borrows. Cereals are harvested at once and stored in accessible places, which makes them easy for an elite to tax. Roots and tubers can stay in the

ground and are harder to seize. The authors even call highly productive root crops a curse of plenty for state building. We did not open Scott's book, so we report it only as the essay describes it.

The essay is also honest about what those works show. Support for the general mechanism is not proof that conquest worked through the same channel.

The uncertain step is the link from easier storage to a larger permanent staff. Rulers had other options. They could trade perishable produce for durable goods, demand labor directly, or leave officials living among farming households. Any of those could erase the grain advantage.

The evidence the claim needs is also rare. Granaries alone do not settle it, since their contents may have fed households or emergency reserves. A big palace does not show how reliably its staff were fed.

One possible falsification test is the following. Within one early state, find annexed districts that supplied quickly perishable food yet supported as many permanent officials and soldiers per resident as grain districts, at similar surpluses and collection costs. That would sink the claim.

The habit of mind here is worth borrowing. In health, as in history, the measure that is easy to count is often not the one that matters. A study can report more of something without showing that the extra can be used.

The same gap appears in the longevity economy. A biomarker that moves is not the same as a year of healthy life gained. The question to ask is the one this essay asks of grain: can the gain actually be captured and put to work?

• Account archives that separate local consumption from shipments to administrative centers. • Archaeology that tracks whether storage and permanent staffing grew together after an annexation. • Cases where states ran on perishable foods, which would test the claim most directly.

Sources

Source 1: https://www.journals.uchicago.edu/doi/abs/10.1086/718372

Read this article online