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THURSDAY, OCTOBER 15, 2026 | VOL 1 NO 13 | ARISING HEALTH

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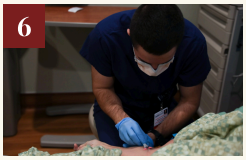
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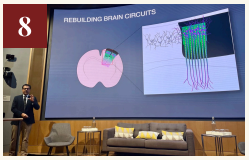
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The Arising

HEALTH INTELLIGENCE

THURSDAY, OCTOBER 15, 2026 · LONGEVITY & HEALTHSPAN DESK

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LONGEVITY & HEALTHSPAN · STANDARD READ · 3 MIN

THE MENOPAUSE SOCIETY

Hormone Therapy Linked to Greater Semaglutide Weight Loss in Midlife Women

Women who took menopause hormone therapy while on semaglutide lost more weight than women who did not, according to The Menopause Society's report of a new study. The researchers also say it is too early to act on the finding.



Photo: “Kalyani Bose lying on a sofa” by xyntia-and-xinnia, CC BY-SA 2.0

This article is based on that report. The study paper itself was not reviewed.

Midlife women are among the heaviest users of GLP-1 weight loss drugs. According to The Menopause Society, about one in five women aged 50 to 64 report current or past use of a GLP-1 obesity medication. That is the highest share of any U.S. demographic group, the society says.

Weight gain is a common worry in this stage of life. The Menopause Society notes that midlife gains are largely driven by aging. Menopause-related factors can also add to fat accumulation and change where fat is stored.

The new study looked at whether hormone therapy changes how women respond to these drugs. According to The Menopause Society's report, it was a retrospective observational cohort study. That means researchers looked back at existing records rather than assigning women to treatments. It included nearly 2,400 women taking semaglutide and nearly 1,500 taking tirzepatide. All were over 45 with a BMI above 27.

Semaglutide acts on one gut hormone pathway, called GLP-1. Tirzepatide acts on that pathway and a second one.

According to the report, the researchers concluded that hormone therapy was associated with significantly greater and sustained weight loss with semaglutide. With tirzepatide, women using hormone therapy initially lost more weight. By the end, the difference did not reach statistical significance. In plain terms, the gap with tirzepatide could not be separated from chance.

The report does not give effect sizes. It does not say how many pounds more women on hormones lost, so the size of the difference is unknown from this source.

Lead author Dr. Regina Castaneda of Mayo Clinic Florida called it a consistent signal. She noted that earlier, smaller studies pointed the same way. She said it is biologically plausible, citing preclinical data that suggest estrogen and GLP-1 signaling may work together. She also said the stronger effect with semaglutide deserves further study.

She was equally clear about the limits. "It is too early to draw firm conclusions," she said, according to the report. She said the findings need confirmation in well-designed prospective trials. She also stressed that weight loss is not an indication for menopause hormone therapy.

That caution matters for personal decisions. An observational study shows an association. Women who use hormone therapy may differ from those who do not in ways the study cannot fully account for. The report does not describe how the researchers handled such differences, and the paper was not available to check. The finding does not show that hormones caused the extra weight loss.

Dr. Stephanie Faubion, medical director of The Menopause Society, said the findings suggest hormone therapy may influence how some women respond to GLP-1-based weight loss medications. She said more research is needed. She added that the study highlights the importance of considering menopause-specific factors when developing personalized approaches to weight management for midlife women.

Our reading of the practical takeaway is narrow, and it is our own inference, not a recommendation from the researchers. Castaneda's caution that weight loss is not an indication for hormone therapy points the same way. Do not start or stop hormone therapy or a GLP-1 medication without talking to your clinician first.

The study will be presented at The Menopause Society's 2026 Annual Meeting in San Diego, October 20-24. The full data, including effect sizes, and prospective trials would be needed to show whether the link holds.

Source: The Menopause Society

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LONGEVITY & HEALTHSPAN · STANDARD READ · 3 MIN

JOURNAL OF CACHEXIA, SARCOPENIA AND MUSCLE · 2 SOURCES

47-Year Swedish Study Finds Aerobic Fitness Starts Declining Around 35

Physical fitness in a general population appears to peak between the mid-20s and mid-30s, then slip a little more each year for decades, according to a Swedish study that tested the same people from age 16 to 63. The study measured aerobic capacity, muscular endurance and leg power. It did not track strength over time: grip strength was measured only at 63. Some news coverage describes the findings as a decline in strength.



Photo: “Emonds Field” by Brick.Ninja, CC BY-SA 2.0

The work is an observational study, published in 2025 in the Journal of Cachexia, Sarcopenia and Muscle. It followed 427 people (48% women), all born in 1958 and randomly selected as a representative sample of Swedish teenagers. Observational means the researchers measured people over time. They did not assign anyone to an exercise programme.

The team, led by Maria Westerståhl at Karolinska Institutet, tested the group at ages 16, 27, 34, 52 and 63. The tests covered aerobic capacity, muscular endurance and leg power. From age 27 on, aerobic capacity was estimated from a cycling test at a moderate effort, not an all-out test. At age 16 it was estimated from a 9-minute run, converted using data from a subgroup who also did the cycle test. Muscular endurance was the number of bench press lifts. Leg power was measured with a jump test.

Estimated aerobic capacity and muscular endurance peaked at ages 26 to 36 in both sexes. After that, they declined gradually. The yearly loss started at 0.3% to 0.6% and rose to 2.0% to 2.5% per year.

For total aerobic capacity, the peak came at 35 in women and 36 in men. The rate of loss was about 0.5% a year at age 40 and about 2% a year at 63. From peak to age 63, the cumulative drop was 30% in women and 33% in men.

Fitness measured per kilogram of body weight peaked earlier in men, at 26, than in women, at 31. From about age 40, the decline was similar in both sexes. The gap between individuals also widened. By 63, the spread in total aerobic capacity was five times larger than at the start.

Most people in the group were not frail at 63. Using standard European cut-offs for weak grip or slow chair rising, no men and four women fell below the line. The authors say the pattern matches what is known from elite athletes. They conclude that a decline in physical capacity can be seen before 40 and can later lead to clinically significant problems, especially in sedentary people.

The news coverage went further on exercise. ScienceDaily, which republished a Karolinska Institutet release, said people who became active in adulthood improved their physical capacity by about 5% to 10%. It also said average performance across measures was 30% to 48% below peak by 63. The portion of the paper available to us confirms the 30% and 33% aerobic figures but does not show the other numbers. Those should be treated as the coverage's account.

The paper does not show that exercise stops the decline. Its physical activity data came from a simple yes-or-no question about leisure-time activity. That cannot capture how hard or how long anyone trained. People with a university degree or an active youth were also more likely to take part. The authors included the whole starting cohort in their models to limit bias from dropouts.

Westerståhl said in the Karolinska Institutet release carried by ScienceDaily: "It is never too late to start moving. Our study shows that physical activity can slow the decline in performance, even if it cannot completely stop it." She added that the team will now look for the mechanisms behind the peak at about 35.

A second paper from the same cohort, in Scientific Reports in 2026, links earlier fitness to artery health. Higher aerobic capacity at 34 and at 52 predicted lower arterial stiffness at 63. The association held after accounting for obesity, smoking, blood pressure and HDL cholesterol. It is also an observational finding, so it does not prove that fitness caused healthier arteries.

Taken together, the studies suggest that fitness in the 30s and 40s matters for later life. The data show when decline begins and how fast it speeds up. They do not show how much exercise is needed to change the course. The researchers' next step is to look for the biology behind the early peak and why activity slows the loss but does not halt it.

Sources: Journal of cachexia, sarcopenia and muscle (2025), ScienceDaily (Karolinska Institutet; Journal of Cachexia, Sarcopenia and Muscle)

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LONGEVITY & HEALTHSPAN · STANDARD READ · 3 MIN NATURE AGING · 2 SOURCES

Splicing Gene SRSF1 Made Aged Cells Look Younger and Is Linked to Longer Mouse Life

Turning up a single gene made aged cells look younger in the lab, and aged mice with the gene switched on had a longer median lifespan. That is the finding from a new study in Nature Aging. The publication type is not stated in the material we had. It is early work in cells and mice, and it is not a treatment for people. The lifespan gain is described as associated with the gene, not proven to be caused by it.

The gene is SRSF1. It helps process RNA, the working copies the body makes from DNA. According to the study's abstract, the researchers first built a "transcriptomic age" predictor in human skin cells called fibroblasts. It reads which genes are active in a cell and estimates how old the cell looks. They then ran a screen, a cDNA overexpression screen, switching on genes in aged cells to see which pushed the cells toward a younger pattern.

SRSF1 was the standout. The abstract says it produced strong youthful gene-activity patterns without the cells losing their identity. That point matters. Earlier approaches to reversing aging in cells, which use proteins called Yamanaka factors, can raise concerns about toxicity and efficacy, the abstract notes, and those concerns limit their use in living animals.

In cell experiments, switching on SRSF1 restored youthful traits. The abstract reports less senescence, which is when cells stop dividing but linger and cause trouble. It also reports less protein-handling stress and less inflammatory signaling.

The mouse result is the headline number, but it comes from small groups. In aged mice with SRSF1 switched on, frailty progressed 44% less, according to the abstract. Median lifespan was 14% longer. The groups held 9 to 10 mice each. The abstract describes the lifespan gain as "associated with" SRSF1 expression, which is careful wording. With groups this small, the results need to be repeated before anyone leans on them. This is the weakest part of the story.

The abstract also says the work implicates SRSF1 in aging in worms as well as mice. The authors call it a "conserved regulator of aging," meaning it appears to play a similar role across species. They describe it as a promising target. Only the abstract was available to us, so we cannot report details such as how the gene was delivered, how long effects lasted or whether any side effects appeared. The abstract also does not list the authors or their institutions, and we could not confirm where the work was done.

The abstract's summary names Plesa, Jung and colleagues as the researchers behind the screen. We could not verify further details about the team, and we are not relying on press commentary for any claims here. Any views researchers hold about what the work means are opinions, not findings.

FOR YOU

What does this mean for personal health? Not much yet. Nothing here shows that people can slow aging by targeting splicing. It does not support splicing-related supplements, and it does not support tests that claim to measure splicing activity. SRSF1 was raised artificially in these experiments. Nothing in the study suggests a pill or a diet can do the same thing safely. Mouse lifespan results also often fail to carry over to people. Do not start or stop any medication, treatment or supplement without talking to your clinician.

The value of the work is in the direction it points. It suggests that resetting how cells process RNA could be a way to push back on aging signs without erasing what a cell is. Whether that holds up is the open question.

Watch for larger mouse studies from independent labs, safety data on raising SRSF1 over time, and any sign of how the gene works. Those steps would come well before any human trial.

Sources: Nature Aging (2026), Nature Aging (publisher page)

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LONGEVITY & HEALTHSPAN · STANDARD READ · 3 MIN NATURE · 2 SOURCES

Scans of 100,000 Patients Link Active Brown Fat to Better Metabolic Health

People who have active brown fat tend to have healthier blood sugar, cholesterol and heart measurements, and fewer cardiovascular and psychiatric diagnoses, according to a large new study in Nature. The finding is a pattern in patient records. It does not show that brown fat protects health, and it does not show that anyone should take a blood pressure drug to get more of it. We reviewed only the study's abstract; the cell and mouse details below come from press coverage, not the paper.



Photo: “radiation scanner” by Muffet, CC BY 2.0

The study, published in Nature in 2026, analysed a cohort of 101,243 patients from a large cancer centre. It looked at existing patient data in people, so it can show associations but not cause and effect. Using a machine learning approach, the researchers semi-automatically measured brown fat activity in about 8,700 of those patients.

Brown fat is different from the white fat most people think of when they think of body fat. White fat stores energy. Brown fat, formally brown adipose tissue or BAT, uses energy to produce heat. The study's authors note that earlier work linked detectable brown fat in humans to a lower prevalence of cardiometabolic diseases, meaning conditions of the heart and metabolism. What was missing was a full picture of the health conditions tied to it, and a fast way to measure its activity in large numbers of people.

This study aimed to fill both gaps. Across all diagnoses, cardiovascular and psychiatric diseases were less prevalent in people with detectable brown fat. Among the patients whose brown fat activity was measured, higher activity was linked to lower odds of cardiometabolic disease and to a healthier distribution of white fat. This was especially true for brown fat in the armpit area, which the abstract calls the axillary depot.

People with brown fat also had more favourable blood sugar measures, lipid profiles (blood fats such as cholesterol), inflammatory markers and heart ultrasound readings, according to the study.

The psychiatric link is the least familiar part. The abstract reports lower rates of psychiatric disease in people with brown fat. News-Medical, covering a Rockefeller University release, says this included lower rates of anxiety and depression, and

describes the link as reported for the first time in this study. First author Nicolás Gómez-Banoy offered one possible explanation in that coverage: "We know that metabolically healthy people tend to have lower incidence of psychiatric diseases, so it could be that having brown fat makes you more metabolically healthy, and this correlates with fewer psychiatric issues." That is a hypothesis. The study does not test it.

The drug finding came from the same records. Use of angiotensin receptor blockers, a class of blood pressure medicine, was positively associated with the presence of brown fat. As a proof of concept, the researchers showed that one of these drugs, losartan, increased brown fat cell activity and whole-body energy expenditure. The abstract does not say in which models this was tested. The News-Medical coverage says it was shown in cultured brown fat cells and in healthy mice, which kept a steady body weight.

Why this is not a reason to start losartan. The link in patients is an association, so it cannot show that the drug produced the brown fat. Losartan is a prescription medicine given for a medical need. The abstract does not report that taking it improves health through brown fat in people. The coverage also says the authors flagged possible downsides of brown fat for future study, including higher rates of osteoporosis and skin disease. It adds that the SSRI antidepressants were associated with dampened brown fat activity. Readers should not start losartan, any other angiotensin receptor blocker, or any drug or supplement to boost brown fat without talking to their clinician, and should not change a prescribed medicine without talking to their doctor.

There are other cautions. The patients were treated at a cancer centre, and they may differ from the wider population. The abstract also gives no size for the associations, so how large the health differences are is not stated there.

The authors present the work as a platform for drug repurposing: using existing, well-studied drugs to activate brown fat. In the Rockefeller University release, republished by News-Medical, Gómez-Banoy said losartan's brown fat effects appear to work through different molecular pathways than its blood pressure effects. If so, future treatments might separate the two. Senior author Paul Cohen said in the same release, "This is just a start." What to watch next is whether studies in people can show that switching on brown fat improves health, and whether the other medications the team flagged hold up.

Sources: Nature (2026), News-Medical (Rockefeller University release; study in Nature)

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LONGEVITY & HEALTHSPAN · STANDARD READ · 2 MIN NEWS-MEDICAL

Late-Night Eating Linked to 20-30% Higher Liver Disease Rates

People who reported eating late at night had 20% to 30% higher rates of liver disease than people who did not, according to News-Medical's report of a new study from Oregon Health & Science University (OHSU).

This article is based on that report. The study paper itself was not reviewed.

According to News-Medical, the researchers define late-night eating as eating after dinner or after bedtime. They looked at two liver conditions: metabolic dysfunction-associated steatotic liver disease (MASLD), which is the newer name for fatty liver disease, and cirrhosis, which is severe scarring of the liver.

News-Medical reports that the team analyzed three large, existing U.S. datasets: the Nurses' Health Study, the Nurses' Health Study II and the Health Professionals Follow-up Study. Almost 200,000 participants had reported their diet and lifestyle habits. The researchers grouped people by eating behavior and looked for links to

reported fatty liver disease or cirrhosis. They found a 20% to 30% increase in all forms of liver disease among late-night eaters compared with those who did not eat late.

The report says earlier research had tied late-night snacking to heart disease and obesity. Lead author Robert Wilechansky, M.D., a hepatologist at OHSU, said this is among the first long-term analyses to explicitly connect it to fatty liver disease. He noted that as much as 30% of the U.S. population is affected by fatty liver disease.

Why might timing matter? Wilechansky said the liver is closely tuned to the body's circadian rhythms, the internal clock that shapes our sleep-wake cycle. Co-author Andrew McHill, Ph.D., a sleep and circadian physiologist at OHSU, said eating at night, when the body clock is promoting sleep, reduces the body's ability to metabolize food efficiently. He said this can eventually lead to obesity, diabetes and, now, poor liver health. McHill also noted that animal research shows the liver to be among the organs most vulnerable to a mismatch between eating and sleep timing.

The findings come with limits. Wilechansky cautioned that the study is observational and relies on broadly defined, self-reported data. That means it shows a link, not that late eating causes liver disease. Other differences between late-night eaters and everyone else could play a role.

The risk also needs context. Wilechansky said that of the people who develop fatty liver disease, only a small fraction go on to serious forms such as cirrhosis. Still, he said the main takeaway is that confining calorie intake to the hours before bedtime might be beneficial. He noted this idea concerns timing, not changing what people eat.

FOR YOU

For readers thinking about healthy aging, the practical point is modest. Wilechansky's suggestion is that confining calories to the hours before bedtime might be beneficial, but the evidence so far does not show that moving meals earlier will protect the liver. Readers with liver disease or diabetes should talk with their clinician before making significant changes to eating patterns.

WHAT TO WATCH NEXT

According to News-Medical, Wilechansky is part of an OHSU team recruiting for a study that will monitor eating behavior directly and test whether eating earlier improves clinical measures of fatty liver disease.

Source: News-Medical (reporting on an Oregon Health & Science University study in Clinical Gastroenterology and Hepatology)

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LONGEVITY & HEALTHSPAN · STANDARD READ · 2 MIN JAMA NEUROLOGY

Alzheimer's Blood Test Tracks Brain Amyloid Similarly Across Racial Groups

A blood test for Alzheimer's disease may work about equally well across racial groups at flagging amyloid buildup in the brain, but the typical level of the marker differs from group to group, which raises a question about whether one cutoff can fit everyone. That cutoff question is our own inference, not a tested result.

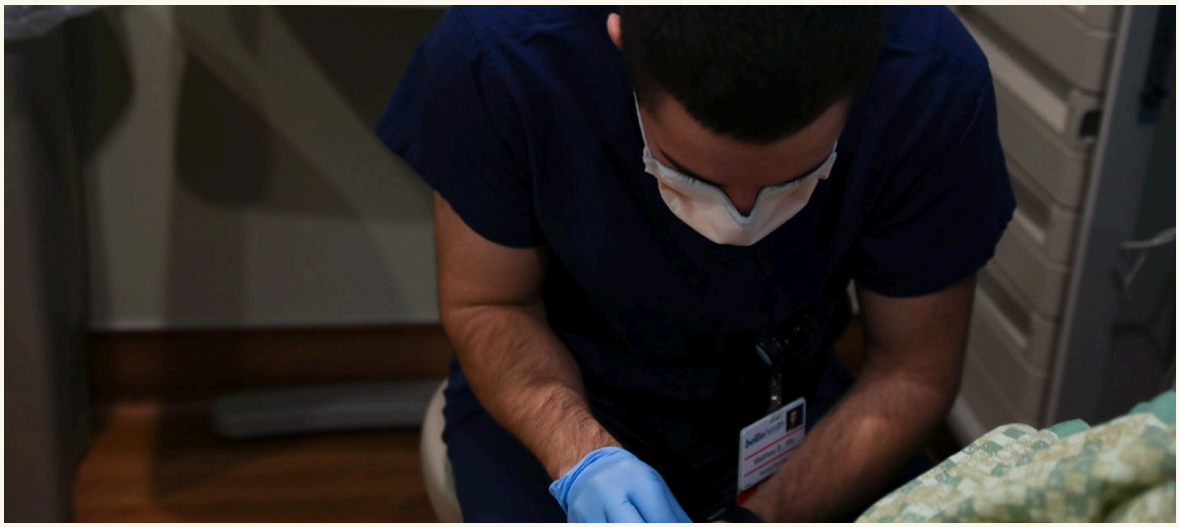


Photo: "22-0001-126 (7013663)" by NavyMedicine, Public domain

The finding comes from a cross-sectional study in JAMA Neurology (2026). That is an observational design: it looks at people at one point in time and can show links, not cause and effect. The researchers analysed screening data from 15,612 adults aged 55 to 80 who were being screened for a trial called AHEAD 3-45. The data were collected from July 2020 to October 2024 at 106 sites across four world regions.

The trial targets preclinical Alzheimer's disease. That means amyloid, a protein that builds up in the brain in Alzheimer's, is present, but there are no cognitive symptoms yet. The blood test measures the percentage of a form of tau protein called p-tau217. The study asked whether that percentage differs by race and ethnicity, and whether its link to thinking scores and to amyloid on brain scans (PET scans) differs too.

Levels did differ. According to the study, %p-tau217 was lower in Asian, Black and Hispanic participants than in White and non-Hispanic participants. Participants were grouped by self-reported race (Asian, Black or White) and ethnicity (Hispanic or non-Hispanic).

Carrying the APOE ε4 gene variant, a known Alzheimer's risk factor, was linked to higher %p-tau217 in every group. The size of that link differed by race and by ethnicity, according to the authors' statistical tests. Of the participants, 5,755 (36.9%) were APOE ε4 carriers.

The link between the blood marker and amyloid on PET scans showed no significant pairwise differences across racial groups. The same was true for the link with a cognitive score called the PACC5. The authors say this consistency supports using a blood test first, with a PET scan to confirm.

Ethnicity told a more complicated story. The links between the blood marker and both the cognitive score and the PET scans differed between Hispanic and non-Hispanic participants. The authors say these ethnicity-related findings need further investigation. The abstract does not explain why the differences appear.

For people without symptoms, the practical meaning is limited. This study does not show that a blood result predicts who will develop dementia. It looked at screening for a trial, not at health outcomes. The abstract also does not give the specific cutoffs used, so it cannot say how many people would be misclassified under a single threshold.

The study's own framing is that levels vary across groups, and that this may influence who is eligible for trials. A fixed cutoff applied to groups with different typical levels could, in principle, treat them differently. That is where the argument is weakest: the abstract does not test group-specific cutoffs or report how any cutoff performed, so the concern remains a reasoned possibility rather than an established problem.

Researchers will need to test whether group-specific or adjusted thresholds improve accuracy, and to follow up the ethnicity findings in further studies.

Source: JAMA Neurology (2026)

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LONGEVITY & HEALTHSPAN · STANDARD READ · 3 MIN CELL · 2 SOURCES

Alzheimer's Gene APOE4 Harms Brain Vessels and Protein Cleanup in Lab Studies

Two new studies from Mount Sinai suggest that APOE4, the strongest genetic risk factor for Alzheimer's disease, does active harm to the brain's blood vessels and its protein cleanup. In one study, blocking a signal called TGF-β reversed the blood vessel damage in the lab; the protein cleanup study showed impairment but no reversal.

The evidence is early. Both papers are laboratory work, not human trials. One, in the journal Cell, combined analysis of human brain tissue data with cell experiments and mouse brain tissue. The other, in Cell Stem Cell, used "miBrains," three-dimensional human brain-like tissue grown from stem cells in a dish. Neither tested a treatment in people. The abstracts available to us do not give sample sizes.

The Cell study first asked why blood vessels in the brain deteriorate in Alzheimer's. The researchers built a single-cell atlas of human brain blood vessels, which maps gene activity cell by cell. In APOE4 carriers, they report, there were fewer pericytes, cells that wrap and support small vessels. A new population of cells that look like myofibroblasts, which produce scar-like tissue, appeared instead. Staining confirmed these cells in APOE4 human and mouse brains.

According to the paper, APOE4 pericytes transform into these myofibroblasts. The new cells secrete a protein called fibronectin, which promotes amyloid buildup in vessel walls. The team identified raised signaling by a molecule called TGF-β as the driver. Blocking TGF-β restored pericyte coverage and reduced vessel scarring and amyloid to the levels seen with the common APOE3 version of the gene.

The Cell Stem Cell study looked at a different problem: how the protein alpha-synuclein piles up. This protein is linked to Parkinson's disease and Lewy body dementia. As in the human brain, miBrains with two copies of APOE4 had more harmful alpha-synuclein. The researchers report that cholesterol accumulated in APOE4 astrocytes, support cells in the brain. That disrupted the cells' waste-disposal compartments, called endolysosomes, so soluble alpha-synuclein was not broken down properly. It then changed into a harmful form that seeded clumps in neurons. The abstract reports no reversal of this process.

Why does cholesterol handling matter here? Because this work points to cholesterol inside brain support cells as a link between the APOE4 gene and failed cleanup. The studies do not examine cholesterol in the blood, diet, or cholesterol-lowering drugs. They offer no basis for changing any of those, and no one should start or stop any medication or supplement without talking to their clinician.

For people who carry APOE4, the practical message is limited. APOE4 raises risk; it is not a diagnosis. These studies describe mechanisms that may explain part of that risk, and they suggest blood vessel damage may be a driver of disease rather than only a late result. Joel Blanchard, the corresponding author, said in Mount Sinai's release, as carried by ScienceDaily, that vessel damage "is a biologically active process caused by APOE4 that may be reversible."

The coverage goes somewhat further than the abstracts. ScienceDaily says the TGF-β result was reproduced in aged APOE4 mice and describes the findings as pointing to treatment targets. The Cell abstract itself says only that the mechanism is "targetable." Blocking TGF-β affects many tissues, and the abstracts say nothing

about safety. A dish or a mouse is a long way from a medicine that works safely in people. The argument is weakest here: reversal was shown only for the vessel findings, and only in the lab.

WHAT TO WATCH NEXT

Whether TGF-β blockers or cholesterol-focused approaches are tested for safety and benefit in APOE4 carriers, and whether other groups reproduce these results.

Sources: Cell (2026), ScienceDaily (Icahn School of Medicine at Mount Sinai release; Cell and Cell Stem Cell)

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LONGEVITY & HEALTHSPAN · STANDARD READ · 2 MIN NATURE · 2 SOURCES

Mouse Study Finds Brain Circuit That Makes Past Trauma Amplify New Stress

People who have been through hard times often react more strongly to the next stressful event. A new study in mice, published in the journal Nature, points to a specific brain pathway that may help explain why.



Photo: “Recovery of brain circuits after stroke or TBI” by jurvetson, CC BY 2.0

This is early animal research. The study was done in mice, not people. The abstract does not state how many animals were used, so we cannot report that figure. Whether the same pathway works this way in humans is not known.

The researchers, led by Zachary Pennington and Denise Cai at the Icahn School of Medicine at Mount Sinai, started with a broad question. They wanted to know which brain circuits are changed by earlier adversity so that later stress hits harder. Instead of checking only familiar stress regions, they used unbiased whole-brain activity mapping, which looks across the whole brain at once.

That search pointed to the anterior hypothalamic nucleus, or AHN. According to the study, this region has received little attention in stress research until now. Mice that had been stressed before showed heightened stress reactivity, and this came with stronger correlated activity between the AHN and a network of brain areas linked to threat.

Next, the team recorded the activity of individual AHN neurons using a miniature microscope. They found that AHN activity scales with negative valence, meaning how unpleasant an experience is. Previous stress increased the share of AHN neurons that were sensitive to valence. The authors say this suggests that inflated processing of negative experiences in the AHN might drive the heightened stress sensitivity.

The study then tested cause and effect. Inhibiting AHN neurons blunted stress responses. Exciting those neurons promoted them. That two-way result supports a causal role for the AHN, at least in these mice.

The team also looked upstream. Neurons in the amygdala, a brain area tied to threat, that send signals to the AHN were found to track negative valence. When the researchers silenced these amygdala inputs to the AHN, the sensitized stress responses disappeared. The authors conclude that the AHN helps regulate negative signals coming from the amygdala, and that this is a new pathway for heightened sensitivity to stressful events.

In comments reported by Neuroscience News, Cai said the circuit acted "almost like a volume knob, amplifying or dampening the impact of a threatening experience." That is her description of the findings, not a measured result. Neuroscience News also said the pathway could become a target for conditions such as post-traumatic stress disorder. The abstract does not make that claim. Pennington himself told the outlet, "we still need to determine the contribution of this pathway to stress sensitivity in humans."

FOR YOU

For readers thinking about their own health, the practical message is modest. The work suggests that the extra reactivity after hard experiences may have a physical basis in specific brain circuits, rather than being a matter of willpower. It does not offer a test, a treatment or a reason to change any care plan today. Mouse circuits often do not carry over neatly to people.

WHAT TO WATCH NEXT

Whether researchers can find the same amygdala-to-AHN pathway in human brains, and whether any way of adjusting it proves safe and useful.

Sources: Nature (2026), Neuroscience News (Icahn School of Medicine at Mount Sinai; Nature)

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LONGEVITY & HEALTHSPAN · STANDARD READ · 3 MIN MBIO · 2 SOURCES

Baby Gut Microbes Linked to Later Visual Brain Signals, Not Proven Lifelong Effect

The bacteria in a baby's gut at 4 months old were linked to how the infant's visual brain was working months later. That is the finding of a study of 194 South African infants, published in mBio in 2025. It is early, observational work, and it makes no claim about lifelong brain health. The bigger idea, that the early gut microbiome shapes brain health across a lifetime, comes from a separate opinion piece in STAT by a program director at a research funder and an advocate. It remains a hypothesis.

The study is an observational, longitudinal study in people. It does not test a treatment, and it cannot prove cause and effect. Researchers followed infants from Gugulethu, a community in Cape Town, as part of the Khula Study. Stool

samples and brain recordings were collected at up to three visits in the first 18 months of life.

The team looked at microbial genes that code for enzymes. These enzymes handle brain-active compounds, according to the paper. The compounds included the neurotransmitters GABA and glutamate, the amino acid tryptophan, and short-chain fatty acids such as acetate and butyrate. The paper says short-chain fatty acids are involved in myelination, the insulation of nerve fibres. Stool samples were sequenced to read the microbes' genes.

Brain development was measured with electroencephalography, or EEG. Infants watched a black-and-white checkerboard that flipped every half second. The EEG picked up the brain's response, called the visual-evoked potential. The authors say it reflects how well visual circuits are balanced and how well they are insulated. They pulled six features from it: the timing and size of three signal peaks.

The main result concerns timing. According to the authors, microbial gene sets from around 4 months of age were strongly associated with the visual signal at around 9 to 14 months. These forward-looking links were more numerous than links between gut genes and brain signals measured at the same visit. The authors say this suggests early microbial metabolism may affect later neural plasticity and development.

That wording is cautious, and it should stay that way. The study shows associations. It does not show that the microbes changed the brain. It measures the gut's genetic potential, not the chemicals actually made. It looks at one brain system, vision, in one community. The abstract does not say whether the findings apply to other groups or whether they relate to health outcomes later in life.

The study matters to a longevity audience because of the question behind it. If early gut function does influence brain development, it would add a biological pathway to a story usually told through genes and upbringing. If it does not, the associations may reflect other shared influences, and gut-focused efforts would be off target. Either answer would change where research money and clinical attention go.

The STAT opinion piece goes further than the study does. Holly Baines of Wellcome Leap, who directs its $50 million FORM program, and advocate George Vradenburg argue the early gut microbiome may be one way experience and environment shape the brain. They link it to possible effects reaching Alzheimer's disease decades later. They write that cesarean birth, antibiotic use, formula feeding and maternal BMI can contribute to a microbiome that develops off course.

The mBio study did not test those exposures or dementia, and the op-ed does not discuss the mBio study. The STAT authors do hedge on their own claims. They write that on autism, causation is not established, and that on dementia the research is only now emerging. They also say they are not arguing against antibiotics, cesarean births or formula. Readers should not start, stop or change any medication, feeding or birth decision without talking to their clinician.

The op-ed's central point is about care, not blame. In their words, "the mother's body and the infant's body are one system. We treat them as two." They call for large studies of the gut-brain axis across the life course. They also want biological markers that could flag vulnerability earlier than behavioral screening, which typically starts between 9 and 30 months in the United States, according to the piece.

For now, the practical message for adults is modest. The idea that early gut microbes shape lifelong brain health remains a hypothesis. The infant data are suggestive, and the dementia link is more speculative still. What to watch next: larger and longer studies that follow infants beyond 18 months, test whether gut chemistry itself changes, and show whether any of this carries into later cognition.

Sources: mBio (2025), STAT (Opinion)

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LONGEVITY & HEALTHSPAN · STANDARD READ · 3 MIN

UNIVERSITY OF WASHINGTON INSTITUTE FOR STEM CELL & REGENERATIVE MEDICINE

UW Team Wins $1 Million to Test Mitochondrial Drug in Older Adults

A University of Washington team has won $1 million to test whether a drug aimed at the cell's energy machinery can help ordinary older adults keep their muscle, memory and immune function. A positive result would suggest that some age-related decline might be treated at the cellular level. A negative one would count against that idea for this drug, though one trial could not settle the question. The drug is approved only for a rare disease, so any use for aging would be off-label.

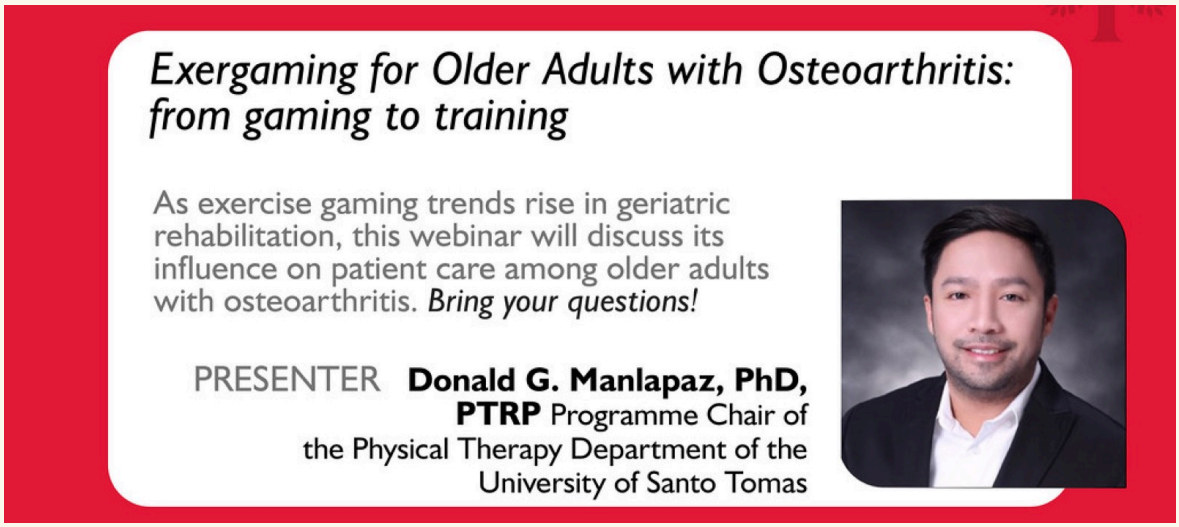


Photo: "ACRM Community Group LIVE Webinar: 22 NOV 2021 (box ad)" by ACRM-Rehabilitation, CC BY-SA 2.0

The award comes from XPRIZE Healthspan, a $101 million global competition launched in November 2023. According to the University of Washington Institute for Stem Cell & Regenerative Medicine, its goal is to develop and clinically test treatments that restore muscle, cognitive and immune performance in older adults by the equivalent of at least 10 years. The stretch goal is 20 years.

The UW group, which calls itself the Mitochondrial All-Stars, learned in August 2026 that it was one of 10 Milestone 2 awardees. It had already received $250,000 in 2025 for a pilot study that, according to the institute, produced encouraging results. The team is led by Dr. David Marcinek, a UW professor of radiology and of laboratory medicine and pathology, working with the company Mighty Therapeutics.

The drug is elamipretide. Mitochondria are the parts of a cell that turn nutrients into ATP, the energy cells use for most of their work. They also help cells handle stress and regulate metabolism and inflammation. Damaged or less efficient mitochondria have been linked to heart and neurodegenerative disease and to biological changes of aging.

Marcinek says elamipretide works through a structural interaction with mitochondria. "Aging leads to a breakdown in the health of the mitochondria," he says. "We think elamipretide helps mitochondria work more efficiently by stabilizing the structure of the membrane, where much of the cell's energy is produced." That is the team's working idea, not a settled finding.

The drug was developed by Stealth BioTherapeutics, now Mighty Therapeutics. In 2025 the FDA granted it accelerated approval for muscle weakness in patients weighing at least 30 kg who have Barth syndrome, a rare inherited disorder that

disrupts mitochondria. That approval does not cover healthy aging. In earlier UW work, Marcinek and Dr. Kevin Conley used magnetic resonance measurements to show that a single dose improved mitochondrial performance in older adults.

The new money will fund a randomized, double-blind, placebo-controlled trial, scheduled to run through 2029. It will test whether elamipretide improves muscle, cognitive and immune function in older adults. Dr. Suman Jayadev, a UW neurologist who cares for patients with brain disease, will help guide the clinical evaluation. That includes blood and other fluid samples, cognitive tests and strength assessments.

Because the work is at an early stage, the team also wants to learn which measures best show how the drug affects health over time. Jayadev says one question is whether the compound could also give a metabolic boost to neurons exposed to risk factors such as amyloid plaque buildup. The weakest point in the case is that the evidence so far is a small pilot and a single-dose measurement, not proof of benefit in healthy aging.

For now, this is a plan to test an idea, not evidence that the drug works in healthy aging. The UW report does not address exercise. As general background not drawn from that report, exercise is the established, widely recommended way to support muscle health. Readers should not start or stop any medication or supplement without consulting their clinician.

Watch for the trial's start and enrollment details, and for any results as the study moves toward 2029.

Source: University of Washington Institute for Stem Cell & Regenerative Medicine

The Arising
HEALTH INTELLIGENCE
THURSDAY, OCTOBER 15, 2026 · MARKETS & STRATEGY DESK

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MARKETS & STRATEGY · STANDARD READ · 2 MIN

REGENERON PRESS RELEASE

Regeneron Plans Trial in Older Adults After Muscle-Sparing Antibody Preserves Lean Mass

Regeneron is betting that the next way to stand out in the crowded GLP-1 market is not more weight loss, but better weight loss, and it has picked older adults as the place to test that idea.

On October 1, 2026, the company reported 52-week results from its Phase 2 COURAGE trial. The trial tested trevogrumab, an antibody that blocks myostatin, alongside semaglutide 2.4 mg in people with obesity. According to Regeneron, the data were presented at the European Association for the Study of Diabetes meeting and are in press with The Lancet.

The numbers come from the lower-dose portion of the trial, with lean mass measured by DXA scan. Over 52 weeks, people on semaglutide plus placebo lost 7.3% of their lean mass. Those who also took 25 mg of trevogrumab lost 5.8%. Those who took 75 mg lost 4.2%, which Regeneron describes as 42.5% preservation relative to

placebo. In an MRI substudy of participants with evaluable data, the lower doses preserved about 69% to 72% of the thigh muscle that semaglutide alone would have cost at 52 weeks.

The drug did not add to the weight loss itself. Regeneron says lower-dose trevogrumab did not meaningfully enhance the weight loss achieved with semaglutide alone. Adverse events were reported by 77% of lower-dose trevogrumab participants and 82% of those on placebo. The most common were nausea, constipation, diarrhea and vomiting.

The strategic signal is in the next step. Regeneron plans a Phase 2 trial of trevogrumab with GLP-1-based therapies in older adults with obesity and decreased muscle mass and/or strength. A prespecified subgroup analysis supports that choice. Participants with low lean mass at the start, defined using the imaging part of the sarcopenia definition from the Foundation for the National Institutes of Health, lost more lean mass on semaglutide alone. They also showed numerically more preservation with trevogrumab.

Regeneron's Boaz Hirshberg said the approach addresses "not the quantity, but rather the quality, of weight loss." The company frames adiposity as the main driver of obesity-related disease, and says its aim is to raise the share of weight lost that is fat.

What this could mean for the market is analysis, not something the release states. A product aimed at older or sarcopenic patients would compete on a different measure than weight-loss totals. It might support a separate patient group, separate pricing arguments and a separate regulatory case. The release says nothing about pricing, trial endpoints or how regulators would view such a claim. It also reports lean mass and muscle volume, not strength or physical function, and it gives no numbers for the higher-dose arm. This is the weakest part of the argument: preserved lean mass has not been shown here to mean better function.

Important limits apply. The results are company-issued. Regeneron states that the safety and efficacy of trevogrumab and olatorepatide have not been evaluated by any regulatory authority. The company also holds rights outside the Chinese Mainland, Hong Kong and Macau to olatorepatide, a GLP-1/GIP receptor agonist that its partner Hansoh has tested in a Phase 3 trial in Chinese patients. Any pairing with trevogrumab would be speculation; the release does not suggest one. Readers should not start, stop or change any medication or treatment, including semaglutide, without talking to their clinician.

Watch for the design of the older-adult trial, especially whether it measures strength and function alongside lean mass, and for the full Lancet publication.

Source: Regeneron press release via BioSpace

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MARKETS & STRATEGY · STANDARD READ · 2 MIN SUNRISE SENIOR LIVING ; BISNOW

BDT & MSD to Buy Sunrise Senior Living From PSP Investments

An investment firm known for hotels, resorts and apartment operators has agreed to take control of one of the best-known names in senior living, and the deal tests whether hospitality skills can win in a care-driven business. The release cites no earlier senior housing holdings for the buyer, but it does not say whether this is its first such investment.

On October 6, 2026, Sunrise Senior Living announced that funds affiliated with BDT & MSD Partners signed a definitive agreement to acquire the company from the Public Sector Pension Investment Board, known as PSP Investments. BDT & MSD will be the majority investor. Sunrise's senior leadership team, led by CEO Jack R. Callison, Jr., will stay in place and invest significant personal capital alongside the buyer. The deal is expected to close in 2027, subject to customary regulatory approvals. The release does not disclose a price.

The asset is large. According to the company, Sunrise operates more than 230 communities in the United States and Canada and serves more than 22,000 residents. Its platform includes a third-party management business, an active joint venture investment platform and a development pipeline of more than 50 new communities. The expected development cost of that pipeline is about $7.5 billion.

The buyer's record is in other property sectors. According to the release, BDT & MSD has invested in or partnered with Greystar, Speed Bay and Auberge Collection. It owns and operates Four Seasons Hualalai, The Boca Raton and Four Seasons Jackson Hole. The release cites no earlier senior housing holdings. It says the firm's hospitality and residential experience gives it a differentiated view of a business where hospitality, customer care, brand and real estate meet.

That framing is the strategic point. Sunrise describes itself as an operator, owner and developer of private-pay luxury and premium communities, and Callison pointed to "lifestyle, wellness, hospitality and care." If a hospitality-minded owner pushes that mix further, rival operators may face pressure on service standards and brand. This is interpretation, not something the release states. Whether it changes competition will depend on results the release does not give.

The seller's view is plain. PSP Investments first invested in Sunrise in 2014 and became sole owner in 2023. Simon Marc, a PSP senior vice president, said the sale "reflects our portfolio rotation strategy, recycling capital from a successfully executed investment into new opportunities." J.P. Morgan Securities and Jones Lang LaSalle Securities advised PSP Investments.

Both buyer and seller point to demographics. The release says generational shifts are driving growing demand for high-quality senior living. Coburn Packard of BDT & MSD said the need is growing and that Sunrise has the leadership and platform to meet it. These are company statements, not independent findings.

Several questions remain open. The release gives no price, financing details or plans for changes in operations. It does not say how the buyer will balance hospitality-style service with the memory care and other specialized services Sunrise also provides. Regulatory review will also shape the timeline, though the release names no specific approvals.

Watch for the closing steps through 2027, any disclosure of terms, and whether other non-specialist investors with hospitality or residential backgrounds make similar moves into senior living.

Source: Sunrise Senior Living (PR Newswire); Bisnow

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MARKETS & STRATEGY · STANDARD READ · 3 MIN STAT

Whoop Moves From Athletes to Medicare Seniors Through Outcome-Based ACCESS Pilot

Whoop built its brand on performance athletes. Now it is asking Medicare to tie its full payment to whether older patients' health measures improve or stay controlled.

According to STAT, Whoop is one of the first consumer wearable companies accepted into Medicare's ACCESS program. The pilot runs for 10 years. It makes the device available at no cost to many traditional Medicare patients ages 65 and up. Whoop is among 55 companies in the program's initial directory, which was published last month.

The payment model is the key change. Through ACCESS, patients get chronic condition management for little or no cost. Medicare pays the companies based on health outcomes. Whoop serves patients with high blood pressure, high cholesterol, obesity and prediabetes. It will provide a wearable, a 12-month app subscription and access to clinicians who can help patients interpret their data.

To receive full payment, Whoop must show it improved or controlled measures tied to these conditions. These include blood pressure, hemoglobin A1C and LDL cholesterol. Chief medical officer Ami Bhatt, speaking at the STAT Summit in Boston,

described the logic this way: "The goal is, how do we bring technology closer to the patient, but not put the cost on the patient — rather, put the demand on the company."

For the market, this shifts risk. A consumer subscription business earns revenue from engagement and retention. Under ACCESS, full payment depends on clinical results. Bhatt acknowledged the gap. Users open the Whoop app about eight times a day, she said. But research on continuous glucose monitors suggests that engagement does not necessarily lead to better health outcomes. Bhatt said Whoop has shown a blood pressure drop of about five millimeters of mercury among its users. STAT's report does not give details of that finding, and it was not described as coming from the ACCESS population.

The product also has to change. Bhatt said simplifying the app is the top item on the roadmap. She cited surveys suggesting that even people older than 80 trust their clinicians with their data. In her words, the issue is how easy it is for them to share it. Whoop is working with the National Council on Aging and AARP on personalizing the product for older adults.

Competitive positioning matters here too. Whoop recently raised $575 million from investors including Mayo Clinic and the sovereign wealth fund of Qatar. Few consumer wearable brands have a Medicare outcomes contract. That could give Whoop a head start in showing payers that its data leads to results. STAT's report does not say how other wearable makers are positioned.

The regulatory line is the main risk. Whoop long sold a product it deliberately kept in the wellness category. The FDA issued a warning letter saying the company's blood pressure-estimating feature qualified as a medical device, not a wellness feature. The agency later closed the letter after Whoop changed the feature, according to STAT. Whoop also announced this year that users can discuss their wearable data with clinicians.

That history shows the tension. Moving toward clinical use brings payment and credibility. It also brings closer scrutiny of what a feature claims to do. A company that measures blood pressure outcomes in a Medicare program will likely face questions about where its wellness features end and its medical claims begin. The source does not say how the FDA would treat future features.

Bhatt also pointed to a practical problem. As a physician, she had patients who brought printed binders of wearable data. A 20-minute visit could not absorb them. She suggested Whoop could send clinicians a contextualized report. It would

combine continuous data with a patient's journal entries and conversations with the app's coach. Readers managing a chronic condition should talk with their clinician before relying on wearable data.

WHAT TO WATCH NEXT

Whether Whoop can show improvements in blood pressure, A1C and LDL cholesterol among Medicare patients, and whether regulators treat its clinician-facing tools as wellness features or medical devices.

Source: STAT

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MARKETS & STRATEGY · STANDARD READ · 2 MIN COMPETITIVE LANDSCAPE · KFF

Typical Medicare Beneficiary Loses Four Advantage Drug Plan Options for 2027

The average Medicare beneficiary will have 28 Medicare Advantage prescription drug plans to choose from in 2027, down from 32 in 2026, according to a KFF analysis published October 1.

That is a loss of four options for the typical person. The count peaked at 36 in 2024. It had grown steadily from 2010 to that point. For 2027, it remains above the 2021 level of 27 and every year before it.

The decline shows up across the wider market too. Counting all Medicare Advantage plans open to general enrollment, with or without drug coverage, the average beneficiary will see 35 plans in 2027, compared with 39 in 2026, according to KFF. KFF's figures exclude special needs plans and employer plans.

The pullback is uneven by state. Texas has the largest drop in drug-plan options for the average beneficiary (nine fewer). Illinois and Ohio each lose eight. Minnesota, New Mexico and Washington each lose seven. Nine states keep the same number as in 2026, and four states gain options, according to KFF. Alaska has none in either year.

Some states remain crowded. Pennsylvania, Michigan and Ohio each offer the average beneficiary at least 40 drug-plan options. In 26 states and Puerto Rico, the average beneficiary has at least 20. At the other end, nine states and the District of Columbia have fewer than 10.

For insurers, the figures point to a market that is trimming overlap rather than leaving. KFF notes that the Centers for Medicare and Medicaid Services (CMS) emphasized stability in the overall marketplace. KFF's data do not say why plans were dropped, so any link to insurer margins, payment rates or regulation would be interpretation, not finding. Still, with the number of options falling in most states, the remaining plans in a county compete against a smaller field.

For beneficiaries, the effect depends on where they live and what plan they hold. KFF says most people whose plan exits live in counties that still have many Medicare Advantage options, plus traditional Medicare. Some can move to another plan from the same insurer. Many will be moved automatically if the insurer has a plan of the same type, such as an HMO or PPO, in the same county. Others must choose a new plan or will default to traditional Medicare.

That puts weight on the enrollment window. Marketing began October 1. Open enrollment runs from October 15 through December 7. Yet KFF reports that most beneficiaries say they do not compare coverage options each year. Earlier focus groups found people overwhelmed by the number of choices. Plans also change premiums, cost sharing, out-of-pocket limits, prior authorization rules, drug lists and provider networks each year.

Most Medicare Advantage enrollees are in plans that include drug coverage, according to KFF. About 4% of 2026 enrollees were in plans without it. Most pay no premium beyond the Part B premium.

WHAT TO WATCH NEXT

CMS releases plan benefit and cost data separately, and KFF says a fuller analysis will follow. Those details will show whether fewer plans also means thinner benefits or tighter rules.

Source: KFF

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CMS Finalizes Narrowed GLOBE Model Benchmarking Part B Drugs to Foreign Prices

Medicare's plan to tie Part B drug rebates to foreign prices just shrank from a sweeping program to a narrow one. According to Covington & Burling, the federal government now projects $440 million in net Part B savings from the GLOBE Model, down from $11.9 billion in the proposal.

The Centers for Medicare & Medicaid Services (CMS) issued the final rule on September 30, 2026. Covington's alert is dated October 2. The model is mandatory for the drugs it covers. Under it, a manufacturer owes a rebate when the U.S. price of a covered drug exceeds a benchmark based on prices abroad. This replaces the usual Part B inflation rebate, which applies when price increases outpace inflation. CMS relies on Section 1115A of the Social Security Act for this change.

The covered drugs are Part B rebatable single-source drugs and sole-source biologics. They must fall in one of seven drug classes and have more than $100 million in annual Part B spending over a 12-month period. The classes are antigout agents, antineoplastics, blood products and modifiers, central nervous system agents, immunological agents, metabolic bone disease agents and ophthalmic

agents. Cancer, bone and eye products used by older patients are squarely in scope. Generics, biosimilars and drugs with a negotiated Medicare price already in effect are exempt.

The benchmark draws on a basket of 19 reference countries, including Canada, Germany, Japan and the United Kingdom. Prices are adjusted for GDP, and the final rule uses World Bank data for that adjustment. Manufacturers can rely on pricing information CMS gathers from third parties (Method I) or submit their own (Method II). CMS clarified that a company may choose Method II even when a licensing partner limits its access to some foreign price data. In that case, the company must explain each missing data element, and CMS will review the explanation.

Two things drive the smaller impact, and the source does not rank them. Covington calls the new exclusions, discussed below, likely one factor. CMS also confirmed that manufacturers that signed Participation Agreements for the GENEROUS Model, its Medicaid pricing model, by August 17, 2026 will be exempt from GLOBE. The exemption applies for each calendar quarter in which the company takes part in GENEROUS. CMS says the reason is that GLOBE could make it harder to isolate GENEROUS effects. Using an illustrative set of drugs, CMS estimates participating manufacturers fall from 19 to 4.

That makes the Medicaid decision a Part B decision as well. Companies that joined GENEROUS by the deadline avoid GLOBE rebates. The few that did not now carry the exposure. Covington does not say which companies fall into which group, and the 19-to-4 figure is based on illustrative drugs, not a final list. Whether the exemption is worth its cost depends on the terms of GENEROUS, which differ materially from GLOBE according to Covington.

The rule also carves out whole product categories. Orphan-only drugs are excluded, which CMS said was done out of caution for beneficiaries with rare diseases who may rely on them. Commenters had warned that the proposal could discourage rare disease research. Certain approved cell and gene therapies on the FDA's list are excluded because of manufacturing complexity. CMS said it may revisit them through future rulemaking. Plasma-derived products are also out. CMS did not state its reasoning, though it had earlier asked whether shortage risk and reduced rebates justified exclusion.

The timeline moved back. The model's start slipped from October 1, 2026 to January 1, 2027. The first performance year begins April 1, 2027, two quarters later than proposed. The performance period runs through March 31, 2032, and the payment

period through March 31, 2034 so that accrued rebates can be paid. Manufacturer data submission runs from January 1 to March 31, 2027.

Beneficiaries may see effects too. Per the rule, cost sharing will be reduced for people in the model cohort who receive a GLOBE drug, and Medicare premiums may fall for people in Part B or Medicare Advantage. Patients should not change treatment because of pricing changes and should talk to their clinician or plan with questions.

For the companies still covered, the benchmark may still matter beyond Medicare. That is an interpretation, not something the source states. The precedent of foreign reference pricing in a mandatory Medicare program is the strategic issue, even at a reduced dollar scale. The weakest part of this analysis is the split between causes of the smaller savings: the source supports both the exclusions and the GENEROUS exemptions as factors but does not say which matters more.

WHAT TO WATCH NEXT

The final rule for the companion GUARD Model, which remains under review at the Office of Management and Budget, and the model's first performance year, which begins April 1, 2027.

Source: Covington & Burling (CMS final rule, Federal Register, Oct. 2, 2026)