



(George Wylesol/For The Washington Post)

7 surprisingly hopeful things we've learned about dementia

These findings — from a blood test to improvements in drug delivery — may lead to better diagnosis and treatment of the memory-robbing condition.

Today at 6:00 a.m. EST

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 By [Richard Sima](#)

If you are like a lot of people, you might be anxious about the risk of getting dementia as you age.

The lifetime risk of developing dementia after age 55 is estimated at 42 percent, according to a [2025 study](#) of over 15,000 participants. The number of Americans developing dementia each year is estimated to increase from 514,000 in 2020 to about [1 million by 2060](#).

But there have been exciting strides in the diagnosis and treatments for Alzheimer's disease, which accounts for 60 to 80 percent of dementia cases, as well as in understanding biological causes and development of dementia more broadly. About half of dementia cases may be

preventable by addressing known risk factors, according to a 2024 [Lancet Commission report](#).

With these advances, it is important to “press down on the gas pedal and really accelerate this work,” said Ronald Petersen, a professor of neurology and the former director of the Alzheimer’s Disease Research Center at the Mayo Clinic College of Medicine and Science.

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We are seeing a “spectrum” of developments from the “low-risk, high public health level all the way through to high-risk, individual intensive therapies,” said Nick Fox, a clinician and professor of neurology at University College London, where he directs the Dementia Research Center.

“I think we’re at the threshold of making a significant impact on the quality of life — the health span, not just the lifespan,” Petersen said.

Here are some of the exciting advances in dementia research and treatments in 2025.

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1. An Alzheimer’s blood test

In May, the Food and Drug Administration approved the first [blood test](#) to detect signals of amyloid beta plaques and tau tangles — the biological hallmarks of Alzheimer’s disease — with over 90 percent accuracy.

“I think this blood biomarker is going to really revolutionize how we diagnose, who can get diagnosed and who’s doing the diagnosis,” said

Kristine Yaffe, a professor and the vice chair of the department of psychiatry at the University of California at San Francisco.

For about a decade, clinicians could measure amyloid beta with PET neuroimaging or indications of plaque formation with a lumbar puncture that collects cerebrospinal fluid. But “PET scans are expensive, and lumbar punctures are invasive,” Petersen said. The new blood test can be administered by a primary care provider and represents what some are calling the “democratizing of Alzheimer’s disease diagnostic tests,” he said.

Experts expect that the blood test will make Alzheimer’s diagnostics more accessible, affordable and available in areas where it would otherwise be difficult to receive a clinical diagnosis because of a lack of medical specialists or equipment.

Around the same time as the blood test approval, the Alzheimer’s Association produced the first diagnostic clinical practice guideline using robust scientific literature assessments and incorporating blood-based biomarker tests, said Heather Snyder, the senior vice president of medical and scientific relations at the association.

The blood test measures two key biomarkers of Alzheimer’s disease. One is amyloid beta, a protein that can misfold and create sticky plaques in the brain. The other is p-tau217, an abnormally modified version of tau protein that can lead to the formation of disruptive tangles.

Many biomarkers have been studied, but “p-tau217 seems to be the most informative with regard to the likelihood of the person having underlying Alzheimer’s disease biology,” Petersen said.

Research shows that the p-tau217 biomarker can serve as a warning sign for Alzheimer’s years in advance.

Earlier detection means more opportunity for earlier treatment and intervention, whether with medications or lifestyle changes. There is no cure for Alzheimer’s disease.

While the buildup of amyloid beta plaques and tau tangles is a hallmark of Alzheimer’s, a positive test does not mean the person necessarily has or will develop Alzheimer’s. (Research has found that more than 20 percent of cognitively unimpaired adults over 65 are amyloid positive.)

Improvements in diagnostics, including the blood test, can also help accelerate research into treatments.

Clinical trials targeting specific biological processes can more precisely enroll patients who have those biological biomarkers, Petersen said.

In the future, just as we get routine tests for cholesterol, we could get a blood test covering different biomarkers to create our unique profile for dementia, which could then be tailored for treatment, he said.

“There are now increasing panels of [testing] multiple proteins at the same time,” Fox said. “So I think while blood-based biomarkers are a huge advance, I don’t think we’re at the end of the story yet with them. I think there will be more refinement and more things to come.”

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2. Improving Alzheimer’s treatment delivery

There are two Alzheimer’s treatments approved by the Food and Drug Administration that target and remove amyloid beta: donanemab and lecanemab. They slow Alzheimer’s progression by about 30 percent, which could help many patients “to win four to six months” of sustained functional independence, Fox said.

However, only a tiny proportion — about 1 in 1,000 — of these drug molecules, delivered intravenously, makes it to the brain because of the blood-brain barrier, Fox said.

“The brain is often referred to as a sort of final frontier, partly because it’s so inaccessible,” he said. As a result, the anti-amyloid drugs need to be given at higher dosages, which can lead to more side effects.

But recent advances may help treatments go where they need to go.

Data presented at Alzheimer’s research conferences in 2025 showed that a drug, trontinemab, using “brainshuttle” technology could cross the

blood-brain barrier and clear amyloid plaques with reduced side effects.

Trontinemab, which is developed by health care company Roche, works by “piggybacking” the anti-amyloid treatment onto a molecule that is naturally transported across the blood-brain barrier, Fox said.

“It’s very exciting because the principle will extend to other therapies,” said Fox, who has consulted for Roche.

3. Gene therapy in the brain

There also has been progress on other types of dementia.

In September, a small trial reported the first successful treatment for Huntington’s disease, a rare and devastating neurodegenerative disease that causes cognitive decline as well as movement disturbances and behavioral disruptions.

Huntington’s disease is caused by a mutation on a specific gene. The experimental treatment involves neurosurgery to directly infuse a gene therapy into affected brain areas, which produces a remarkable slowing of the disease — by 75 percent over three years, according to gene therapy company uniQure.

The study is preliminary, conducted in a small number of patients and not published in a peer-reviewed journal. But “it looks like really the first time we’re seeing a slowing of progression in Huntington’s disease,” said Fox, who was not involved in the trial.

Having similar gene therapies would benefit other dementia patients, said Fox, who works with patients with inherited familial Alzheimer’s disease.

4. Increasing focus on inflammation

While amyloid beta continues to be a target of dementia research, scientists are increasingly investigating the role played by inflammation in increasing dementia risk.

“Alzheimer’s is a complex disease, and it’s likely not going to be a single approach,” Snyder said.

Indeed, a study published in July found that people with the APOE4 gene share many changes to their immune system, which may account

for their susceptibility to not only Alzheimer's but also other neurodegenerative diseases.

Inflammation and immune dysfunction cuts across many different neurodegenerative disorders, including dementia and Parkinson's.

"I think a big push now is on immunomodulation for Alzheimer's and other degenerative diseases," said Yaffe, speaking about ways of modifying immune system activity.

5. Vaccines may reduce dementia risk

One way we could modify immune system activity linked to reduced dementia risk? Vaccines.

Recently, several large-scale studies compared the outcomes of people who received vaccines to those who did not.

Together, they provide robust evidence that vaccines could fight dementia risk.

In April, one study published in Nature tracked more than 280,000 adults in Wales and found that the shingles vaccine cut the risk of developing dementia by 20 percent over a seven-year period. In June, another study tracking more than 430,000 adults found that vaccines against shingles as well as respiratory syncytial virus (RSV) were associated with reduced dementia risk.

And in December, a large follow-up study showed that the shingles vaccine may slow down dementia progression. The shot not only reduced the risk of mild cognitive impairment for people who were cognitively healthy, but also decreased the mortality rate in people who already had dementia.

There are two broad biological hypotheses for why vaccines are linked to reduced dementia risk. First, vaccines could reduce the risk of infections, which have been linked to increased dementia risk. Second, the vaccine itself may activate the immune system in a beneficial way.

These two mechanisms are not mutually exclusive and may both play a role, researchers said.

6. Lifestyle interventions can lead to better cognition

In July, the [largest lifestyle intervention clinical trial](#) in the United States found that simultaneously targeting multiple areas — nutrition, exercise, cognitive training, health monitoring — improved cognitive measures of participants who were at risk of dementia. Participants in the more structured group improved more than those who were self-guided.

The trial, known as U.S. POINTER, was “a big moment” and “culminates decades of research that really informed the intervention,” including a previous [lifestyle intervention trial](#) conducted in Finland, said Snyder, one of the POINTER study’s authors.

What’s important is that “there are ways you can reduce your risk factors for having Alzheimer’s disease and other dementias” and “actually can improve your cognitive aging profile,” said Yaffe, who ran a smaller [trial on personalized risk reduction](#) in 2024.

For example, a [study](#) published in August suggested that people who have a higher genetic risk of developing Alzheimer’s because they carry the APOE4 gene benefit the most from adhering to a Mediterranean diet.

New data presented at an [Alzheimer’s conference](#) in December showed a more detailed picture of how structured lifestyle interventions affect brain health in different subsets of participants of the main study.

Participants in the structured group had improved blood pressure regulation, which is important for proper blood flow to the brain, one study found. Another showed that the intervention reduced respiratory disturbances during sleep.

A neuroimaging study didn’t find differences in brain volume or Alzheimer’s biomarkers, but reported that people more at risk for Alzheimer’s had greater cognitive benefits from the structured lifestyle intervention.

Data collection and analysis is continuing, and the POINTER trial is expected to yield more insights.

7. A newly discovered link to lithium

In August, a study published in *Nature* reported that the metal [lithium may play a protective role in Alzheimer's](#).

“The idea that lithium is neuroprotective has been around for a while,” said Yaffe, who was not involved in the study.

In a healthy brain, lithium helps to maintain the proper functioning of neurons. Lithium carbonate is also used to [treat bipolar disorder](#).

The study, which was conducted in mice, found that amyloid beta plaques trapped lithium, rendering it less effective. And low lithium produced an inflammatory environment in the brain and was marked by accelerated accumulation of amyloid beta plaques and tau tangles.

Researchers reported that small amounts of lithium orotate could reverse the disease and restore brain function, which points to an exciting potential therapy to test in humans.

“I think the scientific rationale is compelling and interesting, but we need to really evaluate it in the clinical trial to see if it might be therapeutically useful,” Petersen said.

The overall progress being made in the field may be changing the “nihilistic approach to dementia,” Fox said.

“We can make a definitive diagnosis in life now. We can deliver therapies to the brain. We can slow diseases,” he said. “We are at the beginning, not at the end of this journey.”

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