SALSANO INSTITUTE · RESEARCH PERSPECTIVE · OCTOBER 2026

Alzheimer’s Disease in 2026: From Clearing Plaques to Changing the Course

A review of the year’s most important advances in treatment, diagnosis and prevention, and what they mean for patients, clinicians and health systems

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Key findings

- **Alzheimer’s is now a treatable disease, though not yet a curable one.** Two approved anti-amyloid antibodies slow early disease, and the field is learning to use them more safely.
- **Diagnosis is moving from the scanner to the blood draw.** In little more than a year the US went from no cleared Alzheimer’s blood tests to four.
- **Tau has become a credible second target.** In July 2026 a tau-lowering therapy produced the first Phase 2 evidence of tangle removal in early Alzheimer’s.
- **Brain-shuttle antibodies and prevention trials** aim to clear amyloid faster, with fewer side effects, and before symptoms begin.
- **Not every hope survives contact with data.** Semaglutide failed to slow established disease, reinforcing that timing and target matter.
- **The future is combination therapy, earlier intervention and equitable access**, supported by lifestyle measures now backed by randomized evidence.

1. A disease at a turning point

For most of the past century, Alzheimer’s disease was diagnosed late and managed only with symptomatic medicines that did nothing to alter its course. That has changed. The 2026 edition of the annual drug-development pipeline review, published in the Alzheimer’s Association’s translational research journal, describes a pipeline growing in size and diversity, and states plainly that Alzheimer’s is no longer untreatable. Eight Phase 3 trials were due to reach their primary completion dates during 2026, alongside 29 Phase 2 studies.

The defining shift is one of timing. Rather than responding once memory loss disrupts daily life, the field is organising around identifying biological risk early, confirming diagnosis quickly and intervening before irreversible damage has accumulated. Every advance reviewed below fits that pattern.

2. Anti-amyloid antibodies: established, and getting better

Lecanemab (Leqembi) and donanemab (Kisunla) remain the foundation of disease-modifying care for early Alzheimer’s. Both remove amyloid plaques and slow clinical decline in mild cognitive impairment and mild dementia, by roughly a quarter to a third over 18 months in their pivotal trials. They act on different forms of amyloid: lecanemab binds soluble aggregates before they become plaques, while donanemab targets a modified, highly aggregated pyroglutamate form.

Their main limitation has been amyloid-related imaging abnormalities (ARIA), swelling or microbleeds in the brain that are usually silent but occasionally serious. Data presented at the Alzheimer’s Association International Conference (AAIC) in London in July 2026 show that this risk can be managed. A slower four-month titration of donanemab cut ARIA by about a third overall, and among people carrying two copies of the APOE4 gene, the highest-risk group, oedema fell from 57% to 24%.

The next generation aims to do better still. Roche's **trontinemab** pairs an anti-amyloid antibody with a "brain shuttle" that rides the transferrin receptor across the blood–brain barrier. In its Phase Ib/IIa study, 91% of patients at the highest dose became amyloid-negative on PET within 28 weeks, with brain oedema in fewer than 5%. Two Phase 3 trials, TRONTIER 1 and 2, are under way in early symptomatic disease, and at AAIC 2026 Roche unveiled **PrevenTRON**, a planned Phase 3 trial in cognitively unimpaired people at high risk of decline. Infusion reactions in the long-term extension (8.6% on the 12-weekly maintenance schedule) show that the safety profile still needs scrutiny.

Prevention is the frontier for all three companies. Lilly's TRAILBLAZER-ALZ 3 is testing donanemab in preclinical Alzheimer's, using a largely decentralised design, with primary completion expected in November 2027. If treating earlier proves more effective, as the underlying biology suggests, the clinical model for Alzheimer's will begin to resemble that for cholesterol and heart disease.

3. Blood tests are transforming diagnosis

Effective therapy is useless without timely, affordable diagnosis, and until recently confirming amyloid required a PET scan or lumbar puncture. In May 2025 the US Food and Drug Administration cleared Fujirebio's Lumipulse plasma test, which uses the ratio of pTau217 to amyloid-β 1-42. Against PET or spinal-fluid testing, 91.7% of positive and 97.3% of negative results were confirmed. Roche's Elecsys pTau181 test followed in 2025 for ruling out disease in primary care, and C2N's PrecivityAD2 was cleared for adults from age 40.

In August 2026 the FDA cleared the Roche–Lilly **Elecsys pTau217** test, the first single-biomarker blood test cleared to both rule in and rule out amyloid pathology. It runs on thousands of laboratory analysers already installed across the US, and Labcorp and Quest have announced national roll-outs. These tests are aids, not standalone diagnoses, and must be read alongside clinical assessment. But they make it realistic, for the first time, to identify Alzheimer's pathology at scale in primary care.

4. Tau: the second front opens

Amyloid starts the disease, but the spread of tau tangles tracks most closely with symptoms. After years of disappointment, 2026 delivered the first convincing clinical signal. At AAIC, Biogen presented the Phase 2 CELIA trial of **diranersen** (BIIB080), an antisense therapy that reduces production of tau protein. The Alzheimer's Association described it as the first Phase 2 evidence that a drug can remove toxic tau tangles in early Alzheimer's, and patients showed cognitive benefit across several measures.

The result comes with caveats. CELIA missed its primary dose-response endpoint, the lowest dose (60 mg every six months) performed best, and episodes of confusion raised a new safety question. Biogen nonetheless intends to advance to registrational trials. Meanwhile Eisai's antibody **etalanetug** lowered a spinal-fluid marker of tangle formation by 62% at three months and 89% at nine months in inherited Alzheimer's, and one landscape analysis counted 189 tau programmes in development. The lesson from AAIC is that tau therapies must be matched to the right form of the protein, the right cellular compartment and the right disease stage.

5. What setbacks teach us

Not every promising idea succeeds. In November 2025 Novo Nordisk reported that oral semaglutide, the GLP-1 drug behind Ozempic and Wegovy, failed to slow progression in the EVOKE and EVOKE+ trials of 3,808 people with early Alzheimer's, despite favourable changes in biomarkers and inflammation. The trials were large and well run, so the answer for established disease is clear. Population data had always pointed toward a *preventive* effect, however, and that question remains open. The broader lesson, that biomarker improvement does not guarantee clinical benefit, applies across the field.

6. New biology worth watching

Laboratory research continues to open new avenues, though all of the following are at an early stage and largely tested in animals. Harvard researchers reported that lithium occurs naturally in the brain, that its depletion is among the earliest changes in Alzheimer's, and that a lithium compound reversed pathology and memory loss in mice. Other groups have identified the brain's immune cells, microglia, as drivers of the sleep loss that may accelerate damage; an enzyme called IDOL whose removal lowered plaques and APOE levels; and a compound acting on GRK2 that slowed nerve-cell loss in mice. These findings broaden the target list; they are not yet treatments.

7. Prevention now has randomized evidence

Some of the year's most consequential findings involve no drug at all. The US POINTER trial showed that a structured programme of physical activity, healthy eating, social engagement and cardiovascular risk management improves cognition in older adults at risk. Twenty-year follow-up of the randomized ACTIVE trial, published in February 2026, found that participants who completed speed-of-processing training with booster sessions had a 25% lower risk of Alzheimer's and related dementia diagnoses. Prevention is no longer only common-sense advice; it is increasingly evidence-based medicine.

8. The challenges that remain

Four problems stand between these advances and the 50-million-plus people living with dementia worldwide. **Benefit is modest:** current antibodies slow decline rather than stop it, so combination approaches across amyloid, tau, inflammation and metabolism are essential, and three quarters of current trials already target pathways beyond amyloid and tau. **Safety requires infrastructure:** MRI monitoring, genetic testing and infusion capacity are scarce outside wealthy health systems. **Cost and access** remain prohibitive in most of the world. And **evidence is uneven:** trial populations still under-represent many ethnic groups and low- and middle-income countries, where the burden of dementia is growing fastest.

9. The Salsano Institute perspective

We believe the coming decade will be defined less by single breakthroughs than by integration: combining therapies, intervening earlier, and making diagnosis and care reachable for everyone. Our view of where effort should concentrate follows from that.

Priorities for the field

- **Combination science.** Rigorous trials pairing amyloid clearance with tau-lowering, anti-inflammatory and metabolic approaches.
- **Equitable early detection.** Validating blood-based biomarkers across diverse populations and in primary care in low- and middle-income settings.
- **AI-enabled research.** Applying machine learning to biomarker, imaging and real-world data to predict who will benefit and who is at risk of side effects.
- **Prevention at scale.** Turning the evidence from POINTER and ACTIVE into accessible public-health programmes.
- **Education as infrastructure.** Free, high-quality training for clinicians and open public education on brain health, because treatments only help people who are diagnosed in time.

Alzheimer's has moved, within a few years, from a diagnosis without options to a disease that can be detected early, slowed and increasingly targeted at its roots. The task now is to ensure that this progress reaches every patient, not only those in a handful of well-resourced health systems.

About the Salsano Institute. The Salsano Institute is an independent think tank dedicated to advancing life science and free access to education. Working alongside leading academic, scientific and philanthropic institutions, it combines independent research with translational insight from partners in life sciences, biotechnology and artificial intelligence. Offices in Washington, D.C. and London.

This paper is for general information and does not constitute medical advice. Treatment decisions should be made with a qualified clinician. Data reflect publicly reported information as of October 2026.

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