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Artificial Intelligence Applications to Neurodegenerative Diseases

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Abstract

Neurodegenerative diseases present fundamental challenges not merely in their biology but also in their modeling. Artificial intelligence (AI) can help reduce that complexity, identify who might remain cognitively normal, who will progress to dementia, and who is likely to benefit from therapy. After introductory remarks on the six stages of application of AI, its real-world tools and systems are described for detecting subtle biological and behavioral changes long before traditional clinical symptoms appear. Approaches to early detection and diagnosis are summarized with their corresponding time frames from brain changes, speech and language patterns, digital behavior, analysis of patients' records, and cognitive micro changes. Precision (or personalized) medicine approach to disease management and care is set forth. AI platforms used to screen thousands of existing FDA-approved drugs to find "hidden" Alzheimer's treatments as well as newly discovered molecules are summarized. AI-led research conducted to find the next generation of biological targets beyond the traditional amyloid and tau proteins is also reviewed.

Abbreviations

AA: Alzheimer's Association; AAIC: Alzheimer's Association International Conference; AD: Alzheimer's disease; AI: Artificial intelligence; ANN: Artificial neural networks; APS: Amyloid probability score; BBB: Blood-brain barrier; CDT: Clock drawing test; CNN: Convolutional neural networks; CPHM: Cox proportional hazards model; CPU: Central processing unit; DRIAD: Drug repurposing in AD; EHR: Electronic health records; FTD: Frontotemporal dementia; GenAI: Generative AI; GPT: Generative pretrained transformer; GPU: Graphic processing unit; JAKI: Janus kinase inhibitors; LBD: Lewy body dementia; LLM: Large language models; MCA: Montreal cognitive assessment; MCI: Mild cognitive impairment; ML: Machine learning; MLM: Multilanguage models; MMSE: Mini-mental state examination; MRI: Magnetic resonance imaging; MS:

Multiple sclerosis; NE: Neighborhood explorer; NETTAG: Network topology-based to (disease-) associated genes; NIA: (U.S.) National Institute on Aging; NLP: Natural language processing; NPT: Neuropsychological test; NSA: Narrative speech analysis; NWT: Naming & word-finding tests; PCA: Principal component analysis; PET: Positron emission tomography; PHGDH: Phosphoglycerate dehydrogenase; RCT: Repetition & comprehension tests; RMSE: Root mean squared error; RNN: Recurrent neural models; SVM: Support vector machine; TBI: Traumatic brain injury; UCLA: University of California, Los Angeles; UCSD: University of California, San Diego; UCSF: University of California, San Francisco); VFT: Verbal fluency tests.

Drugs cited: Bumetanide; Diflunisal; Janus kinase inhibitors; Salsalate; Sildenafil (Viagra).

Molecules newly identified: CAMKK2; DDL-218: DDL-920; DSP-0038: FJMU1887: MARCKS2; ONC-84, PGHDH.

Diseases mentioned: Alzheimer's disease; Anomia; Cancer; Epilepsy; Frontotemporal dementia; Lewy body dementia; Multiple sclerosis; Pulmonary embolism; Stroke; Traumatic brain injury.

Keywords

Artificial intelligence; Alzheimer's disease; Cognitive function; Convolutional neural networks; Dementia; Drug repurposing; Neurodegenerative diseases; Neuronal loss; Precision medicine; Recurrent neural models.

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The terms artificial intelligence (AI) and machine learning (ML) are often used interchangeably, the difference being mostly in complexity with a lot of the methods used for AI being based on ML. ML uses simple models repeatedly while AI typically involves higher computational load (larger amount of computing power and memory required to run the model of interest) and more complex networks such as layers of neural networks that allow a more precise definition of an individual person's disease. AI refers to a system that can simulate and execute the processes of human thinking and learning and make informed decisions. It can be of great help in neurodegenerative diseases (NDD) which present fundamental challenges not merely in their biology but also in their modeling. Of particular interest here, Alzheimer's disease (AD) is incredibly heterogeneous in that it affects people very differently from how quickly symptoms appear to how the disease progresses and which proteins are involved, making it difficult to predict, diagnose, and treat it. Early diagnosis and treatment remain challenging due to clinical heterogeneity and insidious progression. AI could offer excellent solutions by integrating multi-modal data to identify pre-symptomatic biomarkers and stratify high-risk cohorts, improving diagnostic accuracy, and assisting with personalizing treatment and care. It can help reduce the disease's complexity, identify who might remain cognitively normal, who will progress to dementia, and who is likely to benefit from therapy. Furthermore, AI can accelerate drug discovery and development through drug-target identification and predictive modeling of compound efficacy.

After introductory remarks on the six stages of application of AI, its real-world tools and systems already being used (or tested) in hospitals and radiology centers to diagnose AD are described. Such tools and systems can detect subtle biological and behavioral changes long before traditional clinical symptoms appear. Approaches to early detection and diagnosis will subsequently be summarized with their corresponding time frames from brain changes that appear long before symptoms appear to blood biomarkers, speech and language patterns, digital behavior, analysis of patients' records, and cognitive micro changes. The precision (or

personalized) medicine approach to disease management and care will then be set forth. The AI platforms used to screen thousands of existing FDA-approved drugs to find "hidden" Alzheimer's treatments as well as the newly discovered molecules currently in clinical trials will be summarized. Lastly, AI-led research conducted to find the next generation of biological targets beyond the traditional amyloid and tau proteins will also be reviewed.

Introduction

AI is becoming a powerful new tool in improving how AD is detected, especially in its early stages when traditional diagnosis can be difficult. It is shifting the diagnosis from late-stage confirmation to early-stage prediction and detection, which is crucial for slowing progression and improving quality of life. The various learning designs are summarized in Sidebar 1.

AI intervenes in the following six stages: (1) Biomarker analysis; (2) Cognitive and behavioral analysis; (3) Early detection from brain scans; (4) Clinical decision support; (5) Predictive modeling; and (6) Drug development and clinical trials. More specifically:

1. Biomarker analysis: AI can process complex biological data such as blood tests, cerebrospinal fluid (CSF) taps to identify biomarkers associated with AD, including particularly beta-amyloid and tau proteins. This helps move diagnosis away from invasive or expensive procedures toward simpler tests.

2. Cognitive and behavioral monitoring: AI-powered applications and tools can analyze speech patterns (hesitations, pauses, vocabulary decline), typing behavior or smartphone usage, and memory test performance over time. Subtle changes here can signal early cognitive decline.

3. Early detection from brain scans: AI models, especially those utilizing deep learning models (DLM), analyze magnetic resonance imaging (MRI) and positron emission tomography (PET) scans to spot subtle brain changes linked to AD such as shrinkage in memory-related areas (like the hippocampus) and amyloid plaque buildup. These patterns can be detected years before symptoms appear, often earlier than a human radiologist could.

4. Clinical decision support: AI systems can combine clinical notes, laboratory results, and imaging to assist neurologists in making more accurate diagnoses and reduce misdiagnosis with other forms of dementia.

5. Predictive modeling: Using patient history, lifestyle data, and genetics, AI can estimate a person's risk of developing AD. For example, models may incorporate genes like APOE ε4, a known risk factor.

6. Drug development and clinical trials: By analyzing massive data sets, AI helps researchers identify suitable patients for trials faster, track disease progression more precisely, and discover potential drug targets thereby accelerating the search for treatments.

Notwithstanding the above capabilities, certain limitations remain and should be kept in mind, namely, (1) AI does not replace the clinician; (2) Models need large and diverse data sets to avoid bias; (3) Privacy and data security should be ongoing concerns; and (4) Regulatory approval is still evolving in healthcare AI.

Real-world AI tools and systems

The following are some real-world AI tools and systems already being used (or tested) in hospitals and radiology centers to diagnose AD, along with how accurate they are compared to human doctors. Nonetheless, as a cautionary note, it must be emphasized that blind application of any model is dangerous. If the training data is biased, the model will be, too. Focus should be on data preparation – ensuring that models are trained on representative data which have been validated using gold-standard benchmarks and deployed only after thorough validation. If a model works well for one group but not on others, rolling out that model may worsen health disparities. Issues of privacy in the models, not just in the data, must also be kept in mind.

BrainSee

This is so far the only FDA-approved AI platform that turns MRI data into actionable disease insight. It allows doctors to use clinical information and brain imaging to get a quick standardized assessment of a person's risk of progressing to dementia within 5 years. It processes a patient's MRI together with cognitive scores to provide a test score between 0 and 100 (the lower the better) that uniquely helps doctors determine the patients' likelihood of progression from mild cognitive impairment (MCI) to Alzheimer-type dementia. Its reported diagnostic accuracy is up to 90%+ and is, thus, competitive with expert neurologists. This is especially helpful for primary care physicians who may not have dementia-specific expertise. (For more details, refer to Sidebar 2.)

According to BrainSee's developers, 98% of patients report comfort and ease during scans, 92% of users see improved quality of life from personalized recommendations, and 95% of patients feel more informed about their brain health.

NeuroQuant

Using AI, NeuroQuant automatically measures brain volume from MRI scans and detects shrinkage in key regions like the hippocampus. It is often more consistent than human radiologists for measuring subtle volume loss and helps standardize diagnosis rather than replace doctors. What doctors look for most, and focus especially on, are hippocampal volume → memory disorders; ventricular size → indirect sign of brain shrinkage; and changes over time → the most important indicator.

Further, there are important caveats in that the percentiles it provides are not diagnoses. Some people naturally fall low or high and yet are completely healthy. Results must be interpreted alongside symptoms (memory, thinking, mood), a neurological exam, and other imaging findings. (Sidebar 3.)

PrecivityAD (from C2N Diagnostics)

Blood tests like PrecivityAD are part of a major shift in Alzheimer's care. They allow an easier and earlier detection, less invasive than spinal taps, and likely to become more common as treatments improve. This test is employed mainly by neurologists, memory clinics, and specialists to evaluate cognitive decline. It is available in the U.S. but is not yet routine everywhere. It is still considered a relatively new test, and many doctors are more familiar with brain imaging (amyloid PET scans) and spinal fluid testing. Further, insurance coverage can vary, so cost may be a factor.

The test uses AI to analyze blood biomarkers (amyloid beta levels + age factors). Traditionally, confirming AD required PET scans or spinal fluid tests, which are invasive and expensive. PrecivityAD offers a less invasive, more accessible option, to help guide diagnosis and treatment decisions earlier with an accuracy of ~ 85–90% in detecting amyloid pathology. It is comparable to these more expensive imaging methods.

Doctors often use PrecivityAD as part of a bigger picture, alongside cognitive testing, medical history, and brain imaging (if available). It can help decide whether further testing is necessary and whether a patient might benefit from newer AD treatments. (Sidebar 4.)

Cognition tools

Examples of cognition tools include applications developed by groups like the Alzheimer's Association (AA)'s research partners. It analyzes speech pauses, word choice, and story recall. Early studies show it is 70-85% accurate, which is especially promising for early, at-home screening. Speech and cognitive testing are central to diagnosing and tracking AD. These tests look for changes in memory, language, reasoning, and communication—often before severe symptoms appear.

They assess core mental functions and include the mini-mental state examination (MMSE), the Montreal cognitive assessment (MCA), the clock drawing test (CDT), and the more comprehensive neuropsychological testing (NPT).

• **MMSE:** It is one of the most widely used screening tools. It tests orientation (date/place), short-term memory, attention, and language. It is quick (10–15 minutes). Lower scores may indicate cognitive impairment.
• **MCA:** It is more sensitive than MMSE for early-stage AD. It tests executive function, memory, attention, and visuospatial skills. It is often used when MCI is suspected.
• **CDT:** Here, the patient draws a clock with a specific time to evaluate planning, visual-spatial ability, and understanding.
• **NPT:** It is an in-depth evaluation by specialists covering memory, problem-solving, attention, and language. It helps distinguish AD from other conditions.

Speech & language tests

Language impairment is a key early sign of AD. Tests include naming & word-finding tests (NWT), verbal fluency tests (VFT), repetition and comprehension tests (RCT), and narrative speech analysis (NSA).

• **NWT:** Here, patients are shown objects or pictures and asked to name them; difficulty retrieving words (anomia) is common.
• **VFT:** The test measures processing speed and lexical access, for example, naming as many animals as one can in 1 minute.
• **RCT:** It assesses understanding and working memory by repeating sentences or following instructions.
• **NSA:** Clinicians analyze grammar, coherence, and richness of speech by asking patients to describe a picture or tell a story.

In early AD, these tests detect subtle memory lapses, trouble finding words, and reduced verbal fluency. In later stages, they detect disorganized speech, difficulty understanding language, and severe memory and reasoning deficits. They are often combined with brain imaging (MRI, PET scans), blood or CSF biomarkers, and functional assessments (daily living skills).

It is important to note that no single speech or cognitive test can diagnose AD on its own. Doctors use a combination of tests, medical history, and imaging to make a diagnosis.

SPOKE (from UCSF)

SPOKE (Scalable precision medicine-oriented knowledge) is a large biomedical knowledge graph designed to integrate many different types of health and biological data into a single connected system for research and clinical use. It can be thought of as "Google maps for biology and medicine" in which the nodes are things like genes, diseases, drugs, proteins, symptoms and the edges are relationships between them (e.g., "drug treats disease," "gene linked to disease"). It is often described as a "database of databases" because it pulls together data from dozens of biomedical sources (genomics, clinical records, drug databases, etc.) into one unified network. Built from 40+ biomedical databases, it integrates millions of entities and tens of millions of relationships and is continuously updated and expanded.

Modern biomedical data is extremely fragmented. SPOKE's goal is to break data silos and connect disparate information, represent biology as interconnected systems (not isolated facts), and enable discovery of hidden relationships (e.g., new drug uses). By connecting all these pieces, SPOKE lets researchers see how everything in human biology is interconnected rather than isolated. This helps researchers and clinicians understand disease mechanisms, generate new hypotheses, and identify potential treatments or drug repurposing opportunities.

SPOKE supports precision medicine, meaning tailoring treatment to individual patients. For example, it can suggest which drugs might work for a specific patient, link genetic variants to diseases, and help discover new therapeutic targets. It also powers tools like the Neighborhood Explorer (NE), which lets users explore connections around a gene, drug, or disease. For example, it identified patterns and potential molecular targets for therapy. It picked up the well-known association between AD and high cholesterol through a variant form of APOE4 (the apolipoprotein E gene). When combined with genetic databases, it also identified a link between osteoporosis and AD in women through a variant in a lesser-known gene called MS4A6A.

In short, SPOKE is a massive, interconnected biomedical knowledge graph that combines data from many sources to help scientists and doctors discover new insights and enable personalized medicine.

State Viewer (from Mayo Clinic)

This test adds a layer of AI analysis to PET scans to identify brain activity patterns linked to nine different types of dementia from a single scan, often with much higher accuracy than standard clinical workflows interpreted by specialists: History and symptoms; MRI or CT scans (structure of the brain); and PET scans (brain activity).

The challenge is that many dementias look very similar—especially early on—so even experienced doctors can misclassify them. StateViewer is a pattern recognition at scale,
comparing an individual's scan to thousands of known cases—far beyond what a human can recall. It is capable of more precise differentiation by helping distinguish between diseases that are often confused, like AD, Lewy body dementia (LBD) and frontotemporal dementia (FTD).

Instead of just a report, doctors get color-coded brain maps showing why the AI made its suggestion. The tool further reduces variability between radiologists and can speed up interpretation. The key differences from the traditional approach are: Instead of human interpretation alone, StateView augments it with AI; and instead of the clinician's limited experience, it is a database-driven (thousands of cases). In contrast to the clinician's subjective interpretation, it is more standardized. And, as opposed to the slower response in complex cases, it is a faster pattern recognition system. However, it does not replace doctors and is merely a decision-support tool.

Tools like StateViewer are part of a bigger shift toward earlier detection of dementia, more personalized treatment plans, and wider use of AI in radiology and neurology. Over time, it may become more widely available across hospitals and integrated into standard imaging workflows. The bottom line is that whereas traditional diagnosis is skilled but sometimes uncertain, StateViewer offers an AI-enhanced clarity and consistency and, while its access is currently limited, it is expanding.

Merge Healthcare (from Watson Health/Merative)

Watson Health (formerly, a business unit of IBM; now a Francisco Partners' company called Merative) was an early, high-profile attempt to bring AI into healthcare. It was designed to help doctors, hospitals, and researchers make better decisions by analyzing huge amounts of medical data. Its main goals included: Assisting with diagnosis and treatment recommendations; analyzing electronic health records (EHR); supporting cancer care and precision medicine; and improving drug discovery and clinical research. It used AI techniques like natural language processing (NLP) to read medical literature, patient records, and clinical trial data—far more than a human could process quickly—and then generate insights. It showed both the potential and the limitations of AI in complex, real-world medical settings.

What lived on from this all-encompassing program were: Health data analytics platforms to organize and interpret massive data sets (e.g., tools that help hospitals analyze patient outcomes, costs, and performance); clinical decision support (in limited forms) to assist doctors—like flagging drug interactions or suggesting guidelines; and population health management to track trends across groups of patients (e.g., identifying high-risk populations). The emphasis is on data, analytics, and workflow tools such as more particularly "Merge healthcare" imaging for medical imaging and radiology systems.

Aidoc (from Included Health)

Aidoc scans CT images for urgent issues like brain bleeds or pulmonary embolisms and flags critical cases so radiologists review them faster.

Viz.ai (from Included Health)

Viz.ai detects strokes from brain scans in real time and alerts specialists immediately (saving critical minutes).

AI assistance with risk factors

Many modifiable risk factors (like hearing loss, cardiovascular health, or poor sleep) play a role in dementia. AI can help track these through apps or wearables, offering personal insights and nudging people toward healthier behavior (e.g., risk reduction for heart disease or stroke) including lifestyle behaviors modifications (e.g., diet, exercise, stress, sleep, smoking, social interactions) … all areas where AI will have a big impact.

AI approaches to early detection and diagnosis

Currently, getting an accurate diagnosis is a challenge, especially in primary care settings. AI could make it easier by using blood-based biomarkers, cognitive tests, and other data to guide clinicians towards an accurate diagnosis based on the clinical information available to them. AI makes essentially complex multi-layered decisions for a clinician available even if the clinician does not have specialty knowledge about dementia.

AI can detect Alzheimer's in the following ways:

1. Brain changes (10–20 years before symptoms): AI can analyze MRI and PET scans to detect tiny shrinkage in memory-related regions (like the hippocampus) and early buildup of amyloid plaques. These changes can begin a decade or more before memory loss starts.

2. Blood biomarkers (5–15 years early): AI-enhanced blood tests (like PrecivityAD) can detect abnormal amyloid beta ratios and tau protein levels. These signals often appear long before cognitive symptoms.

3. Speech and language patterns (5–10 years early): AI can pick up subtle communication changes such as increased pauses or hesitation, simpler vocabulary, and trouble finding specific words. These are often too subtle for humans to notice casually but measurable by AI.

4. Digital behavior (3–8 years early): By analyzing everyday technology use, AI may detect slower typing speed, increased errors, and changes in how someone navigates apps or completes tasks. These patterns reflect early cognitive decline.

5. Analysis of patients records with ML (up to 7 years early): Top predictors include high cholesterol and osteoporosis, the latter in women only.

6. Cognitive micro-changes (3–6 years early): AI-driven cognitive tests can detect slight declines in short-term memory, reduced attention or problem-solving speed, and even tiny deviations over time can signal risk.

Earlier and more accurate AD diagnosis

AI tools and systems can detect subtle biological and behavioral changes long before traditional clinical symptoms appear through the following:

• **Predictive risk assessment:** AI models can predict AD up to seven years before symptoms manifest by analyzing EHRs for risk factors like high cholesterol or osteoporosis.

• **Advanced imaging analysis:** FDA-approved tools like BrainSee (see above and Sidebar 2) use AI to analyze brain scans and predict the likelihood of disease progression within five years.

• **Speech and language monitoring:** AI algorithms analyze speech patterns, such as vocabulary diversity and pause frequency, to identify early signs of cognitive decline with high accuracy.

• **Pattern recognition:** The Mayo Clinic State Viewer (see above) can identify brain activity with much higher accuracy than standard clinical workflows.

What signs is AI looking for?

Instead of obvious symptoms like severe memory loss, AI focuses on micro-signals, such as:

1. Neurological signals: Brain volume loss (fractions of a percent) and early protein buildup patterns.
2. Language Signals: Examples include less descriptive storytelling, repeating ideas, and reduced sentence complexity.
3. Behavioral signals: Examples are inconsistent routines, small drops in task efficiency, and subtle decision-making changes.

Why early detection matters?

Catching Alzheimer's early can: Enable lifestyle changes (exercise, diet, mental activity); help patients plan ahead; improve chances of benefiting from new treatments; and allowing earlier entry into clinical trials.

However, early detection does not always mean that the person will develop dementia or that symptoms will appear soon. Further, Alzheimer's progression varies widely (some people can remain stable for years) and is mainly useful for risk awareness and intervention.

What can be done after early detection?

The approach follows the acronym: DE4S, where D stands for Diet, E for Exercise, S for Stress, Sleep, Smoking, and Social connections:

1. **Follow a brain-healthy diet:** The most studied approach is the MIND diet in which the focus is leafy greens (spinach, kale), berries, whole grains, and fish and olive oil while limiting red meat, sugar, and processed foods.

2. **Exercise (most powerful intervention):** Strong evidence shows exercise can slow cognitive decline. Regular physical activity improves blood flow to the brain and supports neuron health. The aim is 150 minutes/week (walking, swimming, cycling, strength training 2×/week).

3. **Reduce stress:** Chronic stress damages memory-related brain regions. Reducing it includes meditation or mindfulness, light yoga, time spent outdoors, etc.

4. **Protect sleep:** Deep sleep helps clear amyloid proteins from the brain while poor sleep is strongly linked to Alzheimer's risk. The aim is 7–9 hours per night and keeping a consistent sleep schedule. If sleep conditions like sleep apnea exist, they should be remedied.

5. **Reduce/discontinue smoking:** If applicable.

6. **Stay socially connected:** This includes engaging in regular conversations, participating in group activities or

volunteering, and maintaining close relationships. Isolation increases cognitive decline risk.

Also:

7. **Manage heart & metabolic health:** "What's good for your heart is good for your brain". For this purpose, one should control blood pressure, cholesterol, blood sugar (especially to prevent type 2 diabetes). And

8. **Consider medical options:** Doctors may recommend: Monitoring with cognitive tests, medications (in some early cases), and participation in clinical trials. Organizations like the Alzheimer's Association (AA) can help find trials and support programs.

Studies suggest that combining these habits can lower risk by 30–50% or more and delay onset by years, even decades. Consistency matters more than perfection, and one should not rely on so-called "brain supplements" with little scientific backing, quick fixes or miracle cures, or extreme diets. The bottom line is that early AI detection gives something incredibly valuable (time) and the best response may not be a single treatment—it is a lifestyle strategy that supports brain health from multiple angles (Table 1).

Application	How it helps?	Example tools/sources
Early detection	Predicts disease years before symptoms appear	UCSF prediction model
Imaging	Spots subtle brain atrophy or protein buildup	BrainSee
Speech	Detects language changes as a biomarker	NIA speech analysis
Daily support	Monitors falls and assists with self-care	AI-powered wearables
Drug research	Screens existing drugs for new uses	DRIAD platform

Abbreviations: DRIAD: Drug repurposing in AD; NIA: (U.S.) National Institute on Aging; UCSF: University of California, San Francisco

Table 1: Comparison of AI applications in AD care

Precision medicine approach to disease management and care

AI is significantly impacting the management of AD by enabling earlier detection (see above), personalizing treatment plans, and accelerating the discovery of new therapies (see Table 1 below).

• **Personalized treatment and care:** AI helps transition AD care from a "one-size-fits-all" approach to precision (or personalized) medicine.

• **Tailored therapies:** Multiple language models (MLM) can predict how an individual will respond to specific medications based on their genetic and biomarker profile.

• **Daily monitoring:** AI-powered wearables and home sensors track sleep patterns, physical activity, and walking speed to detect health declines or fall risks in real-time.

• **Cognitive training:** Adaptive AI apps provide personalized brain training exercises that adjust in difficulty based on the user's performance, helping to maintain cognitive function.

Drug discovery and research

AI's capabilities in drug discovery extend beyond simply screening compounds. In fact, AI-based platforms can quickly simulate molecular interactions, assess blood-brain barrier (BBB) permeability, and predict adverse effects, significantly reducing the time and cost traditionally required. While no AI drug

candidates for Alzheimer's have completed the full clinical journey yet, it might only be a matter of time until new solutions are brought to patients.

As of 2026, AI-driven drug discovery for AD has moved from the laboratory to human trials, focusing on both repurposed existing drugs or entirely new molecules, thus drastically reducing the time and cost required to find new treatments.

AI-led drug repurposing

AI platforms like DRIAD (Drug repurposing in AD) and NETTAG (Network topology-based to (disease-) associated genes) use ML to screen thousands of existing FDA-approved drugs to find "hidden" Alzheimer's treatments, significantly shortening development timelines. Examples include:

• **Janus kinase inhibitors (JAKI):** The DRIAD platform has prioritized these anti-inflammatory drugs as high-potential candidates for modifying AD pathology.

• **Sildenafil (Viagra):** A significant association was identified between Sildenafil usage and a reduced likelihood of AD, which has since been validated in laboratory models.

• **P300/CBP inhibitors (Salsalate and Diflunisal):** Identified via AI analysis of insurance claims from 7.2 million patients, these drugs are associated with decreased AD incidence and have shown neuroprotective effects in mice.

• **Bumetanide:** AI modeling suggests this common diuretic may offer a specific treatment path for patients carrying the APOE4 risk gene.

New AI-discovered molecules in clinical trials

Several novel compounds designed or optimized by AI are currently in active clinical testing or late-stage preclinical development (Table 2):

• **DDL-920 & DDL-218:** Identified by the Drug Discovery Laboratory of the University of California, Los Angeles (UCLA), these molecules target memory circuitry and protective proteins like sirtuin 1. DDL-920 specifically aims to restore "gamma oscillations" that are responsible for cognitive processing.

• **DSP-0038:** Developed using Exscientia Centaur's platform, this AI-designed candidate targets serotonin receptors to treat AD-related psychosis and is currently in clinical testing.

• **FJMU1887:** A brain-penetrant small-molecule (Galectin-3 inhibitor) was identified through AI-based screening. Recent research highlights its potential to reduce neuroinflammation and improve cognitive function by disrupting the Gal-3/TREM2 interaction.

• **ONC-841 (phase1/2 clinical trial NCT06509659):** On April 9, 2026, OncoC4 announced a significant milestone in which it begun dosing the first participant after various stages ranging from early Phase 1 trials to promising preclinical animal data in a Phase 2 trial.

This anti-SIGLEC 10 antibody, originally developed for oncology, was identified for its ability to normalize microglial function and clear pathogenic protein aggregates in the brain. Its expression may contribute directly to the pathogenesis of late-onset AD. The trial's primary objective was to evaluate the safety, tolerability, and preliminary efficacy of ONC-841 in early-stage AD.

Preclinical data presented at the Alzheimer's Association International Conference (AAIC) showed that ONC-841 successfully restored the function of microglia (brain immune cells) to clear amyloid plaques and reduce levels of phosphorylated tau in mouse models.

Candidate	AI platform/ lead	Action mechanism	Current stage (early 2026)
Bumetanide	Multiple AI studies	APOE4-specific diuretic	Repurposing validation
DDL-920	UCLA Drug Discovery Laboratory	Memory circuit restorer	Preclinical
DSP-0038	Exscientia (Centaur Chemist)	Serotonin receptor modulator	Clinical testing for Alzheimer psychosis
FJMU-1887	Llama-Gram model	Gelectin-inhibitor	Preclinical/lead compound
ONC-841	OncoC4	o Normalization of microglia (the brain's resident immune cells) normalization o Targets SIGLEC 10, an immune regulation protein	Phase 1/2 clinical trial

Table 2: New AI-discovered molecules in clinical trials

AI-inspired research

• **Protein folding:** Tools like Google's DeepMind's AlphaFold have identified the 3D structures of nearly all human proteins, allowing researchers to design drugs that fit specifically into targets related to AD's pathology.

• **Clinical trial optimization:** AI identifies the best candidates for clinical trials by predicting who is most likely to progress or respond to a treatment, making trials faster and more likely to succeed.

AI-identified next generation biological targets

Researchers are also using AI to find the "next generation" of biological targets beyond traditional amyloid and tau:

In silico medicine targets

Using its PandaOmics model, In silico identified targets like MARCKS and CAMKK2 proteins that form abnormal aggregates and represent new pathways for therapeutic intervention.

PHGDH inhibitors for spontaneous AD

About one in nine people aged 65 and older has AD, the most common cause of dementia. While some mutated genes can lead to AD, that connection only accounts for a small percentage of all Alzheimer's patients. Most patients do not have a mutation in a known disease-causing gene; instead, they have "spontaneous" Alzheimer's, and the causes for that are unclear. Discovering those causes could ultimately improve medical care.

In a new (April 23, 2026) AI-aided study, researchers at the University of California, San Diego (UCSD) found that a gene recently recognized as a biomarker for spontaneous AD may be one mechanism of it, due to its previously unknown secondary function. It may lead to a potential treatment against proteins' buildup that obstructs the gene's moonlighting role. This gene (phosphoglycerate dehydrogenase, PHGDH) had previously been discovered as a potential blood biomarker for early detection of AD. Expression levels of the PHGDH gene directly correlate with changes in the brain in AD in that the higher the levels of protein

and RNA produced by the PHGDH gene, the more advanced the disease. That correlation has since been verified in multiple cohorts from different medical centers.

Using mice and human brain organoids, the researchers found that altering the amounts of PHGDH expression had consequential effects on AD. Lower levels corresponded to less disease progression, whereas increasing the levels led to more disease advancement, triggering a pathway that disrupts how cells in the brain turn genes on and off. In other words, PHGDH has a previously unknown role, independent of its enzymatic function, that through a novel pathway leads to spontaneous AD, i.e. PHGDH is indeed a causal gene to spontaneous AD. While everyone has the PHGDH gene, the difference comes down to the expression level of the gene, or how many proteins are made by it.

It should be emphasized that while many current treatments focus on treating the abnormal buildup of the sticky protein called beta-amyloid in the brain, some studies suggest that treating those plaques may be ineffective: essentially, by that stage of accumulation, treatment is too late. (Alternatively, as opined by this author, those plaques may only be symptoms of AD, not its root cause which was suggested to be a brain autoimmune disease having gone rogue.) By contrast, the PGHDH pathway is upstream, so preventing this pathway can reduce amyloid plaque formation in the first place. The researchers identified NCT-503 as a candidate that can penetrate the BBB to inhibit the regulatory role of the PHGDH enzyme, which may drive disease progression.

How do AI and doctors compare?

Compared to doctors, AI performs better in: Detecting tiny patterns in imaging that humans may miss; handling large datasets quickly; and providing consistent, objective measurements. However, clinicians still lead in interpreting context (medical history, behavior, environment), handling ambiguous or complex cases, and communicating diagnosis and care decisions.

The most effective systems today combine AI analysis with neurologist expertise and cognitive testing. This hybrid approach can reach over 90% diagnostic accuracy, especially in the early stages of the disease.

In sum, AI is not replacing doctors but is making Alzheimer's diagnosis earlier, cheaper, and more accurate. In the future, AI models will use simple voice recordings for early screening, routine blood tests will replace expensive scans, and continuous monitoring will be performed via smartphones and wearables.

Conclusions and take-aways

- AI is becoming a powerful new tool in improving how AD is detected, especially in its early stages when traditional diagnosis can be difficult. It is shifting the diagnosis from late-stage confirmation to early-stage prediction and detection, which is crucial for slowing progression and improving quality of life.

- AI intervenes in six stages: Biomarker analysis; cognitive and behavioral analysis; early detection from brain scans; clinical decision support; predictive modeling; and drug development and clinical trials.

- Remaining limitations include: AI does not replace the clinician; models need large and diverse data sets to avoid bias; privacy and data security should be ongoing concerns; and regulatory approval is still evolving in healthcare AI.

- Real-world AI tools and systems already being

used (or tested) in hospitals and radiology centers to diagnose AD include: BrainSee (so far the only FDA-approved AI platform) that turns MRI data into actionable disease insight, helping doctors determine the patients' likelihood of progression from mild cognitive impairment to Alzheimer-type dementia with a reported diagnostic accuracy of up to 90%+; NeuroQuant which automatically measures brain volume from MRI scans and detects shrinkage in the hippocampus and the ventricles. PrecivityAD which analyzes blood biomarkers in a less invasive, more accessible option, to help guide diagnosis and treatment decisions earlier; cognition tools; speech & language tests; SPOKE which is a large biomedical knowledge graph designed to integrate many different types of health and biological data into a single connected system for research and clinical use; State Viewer which adds a layer of AI analysis to PET scans to identify brain activity patterns linked to nine different types of dementia from a single scan; and others.

- AI can detect Alzheimer′s in several ways: Brain changes (10−20 years before symptoms); blood biomarkers (5−15 years early); speech and language patterns (5−10 years early); digital behavior (3−8 years early); analysis of patients′ records (up to 7 years early); and cognitive micro-changes (3–6 years early).

- AI tools and systems can detect subtle biological and behavioral changes long before traditional clinical symptoms appear through predictive risk assessment, advanced imaging analysis, speech and language monitoring; and pattern recognition.

- AI looks for neurological, language, and behavioral signals.
- Early detection matters because it can enable lifestyle changes (exercise, diet, mental activity); help patients plan ahead; improve chances of benefiting from new treatments; and allowing earlier entry into clinical trials. However, early detection does not always mean that the person will develop dementia or that symptoms will appear soon.

- After early detection, the DE4S is recommended, where :D stands for a brain-healthy diet such as the MIND diet; E for exercise, which is the most powerful intervention; S for stress reduction as chronic stress damages memory-related brain regions; S for sleep protection as deep sleep helps clear amyloid proteins from the brain; S for smoking which should be reduced or preferably discontinued; and S for social connections that should be nurtured. In addition, heart and metabolic health should be cared for and existing medical conditions attended to. Combining these habits can lower risk by 30–50% or more and delay onset by years, even decades.

- AI is significantly impacting the management of AD by enabling earlier detection, personalizing treatment and care, cognitive training, and accelerating the discovery of new therapies.

- AI′s capabilities in drug discovery extend beyond simply screening compounds. AI-

driven drug discovery for Alzheimer′s has moved from the laboratory to human trials, focusing on both repurposed existing drugs and discovering entirely new molecules, thus drastically reducing the time and cost required to find new treatments.

- Alzheimer′s diagnosis earlier, cheaper, and more accurate. In the future, AI models will use simple voice recordings for early screening, routine blood tests will replace expensive scans, and continuous monitoring will be performed via smartphones and wearables.

- While Leqembi and Kisunla are the first FDA-approved drugs to directly attack the underlying mechanism of Alzheimer′s, more treatments like them are in the pipeline, and pill-based therapies are on the horizon. Research continues into non-pharmacological treatments for AD, including non-invasive therapies like brain stimulation and focused ultrasound.

- AI is not replacing doctors but is making

Sidebar 1 – AI languages designs

The details of the several languages designs are summarized in the following Table 3:

Method	Design	Details
Machine learning (ML)	A collection of data analysis techniques that aim to generate predictive models by learning from data and improving their ability to make predictions through experience	o ML models are considered shallow learners, working on data with hand-crafted features defined through expert-based knowledge. Raw data must be pre-processed before constructing a ML system, requiring domain expertise to proceed with feature extraction and engineering to train the algorithm appropriately. o Example of ML: SVM accomplishes the classification task by finding the hyperplane that, in the multi-dimensional feature space, optimally separates the data into two (or more) classes
Deep learning (DL)	A sub-field of ML that uses methods that can learn relationships between inputs and outputs by modeling highly non-linear interactions	o DL models are different from shallow learners and can elaborate raw data, thus requiring little or no feature engineering, thanks to their ability to model complex functions and identify relevant aspects in the data distribution. o DL algorithms are based on ANNs) which are inspired by the human brain and can model very complex functions, identifying important aspects in the features and suppressing irrelevant ones o Example of DL: A CNN is

		composed of nodes organized into layers. It can take an image as input, elaborate the features of the image through its layers, and assign a class attribution as output, thus differentiating between two or more groups
Supervised learning	A ML task defined using labeled data sets for algorithm training	o The algorithms learn to give the right answer, as defined by the ground truth set, which has labels assigned to the data. o Example: A SVM performing the classification task
Unsupervised learning	A set of algorithms aimed to discover hidden patterns or data groupings without the need for human intervention	o In unsupervised learning, unlike supervised learning, there are no correct answers and the algorithm's aim is to discover structures within variables. The algorithms work with unlabeled data o Examples: Clustering algorithms and PCA
Classification task	The algorithm is trained to predict a class label	o The algorithm learns to associate input data with an output label in a supervised manner, and its results can be evaluated by metrics such as accuracy score o Example: The classification of patients affected by a disease vs. normal controls
Regression task	The algorithm is trained to predict the value of a continuous variable	o The algorithm learns to associate input data with an output value in a supervised manner, and its results can be evaluated by metrics such as the RMSE o Example: The prediction of hippocampus volume as a numerical quantity
Clustering	Clustering consists of partitioning a data set to find a grouping of the data points.	o Clustering is one of the most important unsupervised learning techniques. Its main goal is to reveal sub-groups within heterogeneous data, in such a way that greater homogeneity is shown within clusters (rather than between clusters) o Clustering algorithms can lead to the identification of patterns across subjects or patients that are difficult to find even for an expert clinician
Overfitting	Overfitting occurs when the model is too dependent on training data to make accurate predictions on test data	When a model is overfitted, its learned ability to separate between two classes does not generalize well to data it has never seen before, therefore limiting its usability for real-world applications
Ensemble learning	Model ensembling consists of combining multiple ML models to obtain better predictive performance than any of the	A single model alone can be weak in generating predictions. Combining multiple models can compensate for their individual

	constituents alone	weaknesses
Transfer learning	A supervised learning technique in which the knowledge previously acquired from the model in one task is used to solve related ones	In a transfer learning approach, the model is first pre-trained on a source task, then re-trained and tested on a target task. The source task should be related to the target task, with similar relations between the input and output data. In fact, in the pre-training phase, the model gains helpful knowledge for the target task.
Cox regression	CPHM is a regression technique for investigating the association between the time of an event occurring and one or more predictor variables	Cox regression gives hazard rates as measures of how factors influence the risk for an event occurrence (outcome), be it death or infection

Source: Fabrizio C et al. (2021)

Abbreviations: ANN: Artificial neural networks; CNN: Convolutional neural network; CPHM: Cox proportional hazards model; PCA: Principal component analysis; RMSE: Root mean squared error; SVM: Support vector machine.

Table 3: Comparison of AI languages designs

Sidebar 2 – The BrainSee test

This FDA-approved, AI-based software test analyzes brain data to predict whether someone with memory problems is likely to develop Alzheimer's dementia in the future. It is mainly used in people with MCI (a condition where someone has noticeable memory or thinking problems but not full dementia).

What is it?

The test helps answer the following two important questions: Will this person stay stable? Or is this person likely to progress to AD within ~5 years? As such, it is one of the first tools focused on forecasting AD, is most useful when one is in the "uncertain middle" stage (MCI) progression, and it works best not alone but when combined with other tests.

How it works?

BrainSee combines: A standard brain MRI scan, cognitive test scores (memory and thinking tests), and AI analysis of brain patterns to produce a score from 0–100: Low score (<30) → low risk; mid-range score (30–70) → moderate risk, and high score (>70) → higher risk of developing AD.

How reliable is it?

Clinical studies of BrainSee report ~90% accuracy in predicting whether someone with MCI will develop AD within about 5 years. Its performance is consistent across different hospitals and MRI scanners, and it works using real-world clinical data, not just ideal lab conditions.

In practice, this means that it is strong for a prediction tool, especially compared to older methods, but it is not perfect, i.e., some people flagged "high risk" will not progress, and some "low risk" people might. In sum, it is useful for risk estimation but is not a definitive answer.

Why is this test different from others?

The test is non-invasive (no injections or spinal taps); it uses tests many patients already get (MRI + memory tests); and it focuses on prediction (prognosis), not just diagnosis. However, it is important to know that the test does not diagnose AD by itself and is used alongside a doctor's evaluation and other tests.

How does it compare to other AD tests?

- **Amyloid PET scan:** It directly measures disease biology, detects amyloid plaques (a hallmark of AD), and is very accurate for diagnosis. However, it is expensive, involves radioactive tracers, and does not predict progression very well. Whereas PET reveals the underlying pathology, BrainSee will predict whether the condition will worsen.
- **Cerebrospinal fluid (CSF) test:** It measures amyloid and tau proteins via spinal tap. It is highly sensitive but invasive and less widely accepted by patients. Whereas CSF provides biological confirmation, BrainSee makes a non-invasive risk prediction.
- **Blood test (newer):** It measures biomarkers like amyloid or tau in the blood. It is simple and convenient and rapidly improving. However, while still improving, it is less predictive for long-term progression. Whereas a blood test provides screening/early detection, BrainSee adds prognosis.
- **Standard cognitive tests:** These are memory and thinking examinations. They are cheap and widely available. However, they cannot predict future decline reliably. Whereas cognitive tests measure current performance, BrainSee predicts a future risk.

Who should consider BrainSee?

- Good candidates for this test are people who: Have MCI, are experiencing persistent memory problems, want to know their risk of developing Alzheimer's, or are deciding about early treatment, lifestyle changes, or clinical trial participation. It is less useful for people with no symptoms, those already diagnosed with advanced AD, or in situations where MRI is not available or feasible.

In the big picture, each test answers a different question: Cognitive tests determine how a person is doing now, cognitively speaking. PET/CSF/blood tests determine whether Alzheimer's pathology is present, and BrainSee predicts how likely is a person to decline cognitively.

Sidebar 3 – The NeuroQuant test

NeuroQuant is a specialized medical software used with brain MRI scans to give precise, quantitative measurements of brain structures. It is especially helpful for tracking changes over multiple MRIs, catching early subtle decline, and providing objective data when symptoms are unclear.

What is it?

NeuroQuant is an FDA-cleared imaging analysis tool that takes a standard MRI of the brain and uses software to measure the size (volume) of different brain regions. Instead of a radiologist just looking at the scan, NeuroQuant provides objective numbers.

How it works?

The images obtained with a regular brain MRI are processed to identify brain regions (like the hippocampus, cortex, ventricles), measures their volumes, and compares these measurements to a normal database (based on age, sex, head size). NeuroQuant then generates a report showing whether areas are normal, larger or smaller than expected. This helps

doctors detect or monitor conditions where brain volume changes matter, such as: AD and dementia, MCI (memory issues), traumatic brain injury (TBI), multiple sclerosis (MS), or epilepsy.

NeuroQuant is especially useful for spotting subtle brain shrinkage (atrophy) that might not be obvious on a regular MRI. Further, it provides objective data instead of just visual interpretation, can detect early or subtle changes, helps track disease progression over time, and assists in treatment decisions. However, it is not a diagnosis by itself but a tool that supports a doctor's evaluation, alongside symptoms, exams, and other tests.

What does the report show?

A typical report includes volumes (usually in cubic millimeters) of brain structures such as the hippocampus (important for memory), amygdala (emotion processing), lateral ventricles (fluid-filled spaces), and whole brain volume. Measurements are reported in percentiles indicating how each structure is compared to a normal population of the same age and sex: 50th percentile: average; above 50th: larger than average; below 50th: smaller than average.

How common patterns are interpreted?

This is best explained by an example, e.g., hippocampus at 12th percentile means smaller than ~88% of people of the same age; ventricle at 90th percentile means larger than most people (can also suggest brain tissue loss nearby). Possible concerns depend on symptoms such as structures below 10th percentile or mild asymmetry (one side smaller than the other). More concerning findings are for very low percentiles (e.g., <5th percentile) in key regions. Patterns like low hippocampal volume may be linked to memory disorders; enlarged ventricles can indicate brain atrophy; and multiple regions shrinking could suggest neurodegeneration.

Limitation

The percentile indicated by the BrainSee report does not confirm a disease – it just raises a flag for the doctor to evaluate further.

The PrecivityAD test

PrecivityAD is a specialized blood test used to help detect AD.

What is it?

PrecivityAD is designed for people (typically older adults) who already show signs of memory problems or cognitive decline. It helps doctors estimate whether those symptoms are likely due to AD.

How it works?

Instead of brain scans or spinal taps, this test uses a simple blood sample to measure specific biomarkers linked to AD: Amyloid beta proteins (Aβ42 and Aβ40) and Apolipoprotein E (APOE) genotype (a genetic risk factor). The results are combined into an amyloid probability score (APS), which estimates the likelihood that a person has amyloid plaques in the brain—a key feature of AD.

What does the report show?

The PrecivityAD report shows the APS as: Low score → unlikely to have amyloid plaques; Intermediate → unclear; more testing needed; High score → likely to have amyloid plaques. The test has shown pretty strong performance, but it is not perfect. Studies report about 85–90% accuracy in predicting whether someone has amyloid plaques (compared to PET scans or spinal fluid tests). It is especially good at ruling out AD when the score is low, identifying high likelihood cases when the score is high, but is less helpful in the intermediate

score wherein patients need to undergo additional testing. In short: it is reliable as a support tool, but not definitive on its own.

Limitations

The test is not a standalone diagnosis as doctors must interpret it along with other exams, imaging, and other tests. Further, it is not meant for general screening (only for people already showing symptoms) and, like all tests, it can have false positives or negatives.

The pTau217 test

This newer blood test was reported on April 14, 2026. It looks for levels of plasma phosphorylated tau 217 (pTau217), a form of tau protein. Tau proteins are known to form toxic tangles in the brains of AD, typically later in their decline. It can reveal amyloid accumulation in the brain farther than 10 to 20 years before symptoms appear, well before clear abnormalities appear on amyloid PET scans. People with low pTau217 are very unlikely to show the development of any plaques or tangles in their brains during later scans. Even when amyloid scans appear normal in the clinic, the pTau217 biomarker can identify individuals who later become amyloid-positive. It also shows that those with low pTau217 levels are likely to stay amyloid-negative for several years.

If the test is validated through further studies, it could help doctors diagnose Alzheimer's risk earlier. Further, as the new Alzheimer's drugs depend on early detection, they do not work for patients in advanced decline.

It still is too early to recommend the blood test as a regular part of screening for seniors, but it might be a good tool for clinical trials testing new AD drugs by identifying people at higher risk. Eventually, this sort of test could be used for regular health maintenance.

Table 4 compares several of the available neurological tests.

Test	For whom?	What is measured?	Purpose
BrainSee	People with MCI or thinking problems	Standard MRI brain images + test scores	o Likelihood to develop AD within ~ 5 years o ~ 90% accuracy
NeuroQuant	People with unclear symptoms	Precise quantitative measurements of volume of hippocampus, cortex, ventricles	o Tracking disease progression over time (atrophy) o Compares with normal data base
PrecivicityAD	Older adults who show signs of memory problems or cognitive decline	Specialized blood test to help detect AD to measure to measure amyloid-beta proteins (Aβ42 and Aβ40) and Apolipoprotein E (APOE) genotype	o 85-90% accuracy o Less helpful in the intermediate score wherein patients need to undergo additional testing
pTau217 test	May be recommended as a screen test for seniors	Blood test to measure amyloid accumulation in the brain 10-20 years before symptoms	Good tool for clinical trials testing new AD drugs by identifying people at higher risk.

Table 4: Comparison of available neurological tests

References

1. Abadir P, Oh E, Chellappa R, Choudhry N, Demiris G, Ganesan D, Karlawish J, Marlin B, Li RM, Dehak N, Arbaje A, Unberath M, Cudjoe T, Chute C, Moore JH, Phan P, Samus Q, Schoenborn NL, Battle A, and Walston JD (2024). "Artificial intelligence and technology collaboratories: Innovating aging research and Alzheimer's care". Alzheimer's Dementia 20(4):3074-9. doi: 10.1002/alz.13710.
2. Aditya Shastry K and Sanjay H (2024). "Artificial intelligence techniques for the effective diagnosis of Alzheimer's disease: A review." Multimed. Tools Appl. 83:40057–92. doi:10.1007/s11042-023-16928-z.
3. Alzheimer's Association (2025). "Earlier diagnosis". https://www.alz.org/alzheimers-dementia/research-and-progress/earlier-diagnosis.
4. Angelucci F, et al. (2024). "Integrating AI in fighting advancing Alzheimer: Diagnosis, prevention, treatment, monitoring, mechanisms, and clinical trials." Current Opinion in Structural Biology. doi:10.1016/j.sbi.2024.102857.
5. Babu B, Parvathy G, Bawa FSM, Gill GS, Patel J, Sibia DS, Sureddi J, and Patel V (2024). "Comparing the artificial intelligence detection models to standard diagnostic methods and alternative models in identifying Alzheimer's disease in at-risk or early symptomatic individuals: A scoping review" Cureus 16(12): e75389. doi: 10.7759/cureus.75389.
6. Barnard L, et al. (2025). "An FDG-PET-based machine learning framework to support neurologic decision-making in Alzheimer's disease and related disorders." Neurology 2025; doi:10.1212/WNL.0000000000213831.
7. Bazarbekov I, Razaque A, Ipalakova M, Yoo J. Assipova Z, and Almisreb AA (2024). "Review of artificial intelligence methods for Alzheimer's disease diagnosis: Insights from neuroimaging to sensor data analysis." Biomed. Signal Proc. Control 92:106023. doi:10.1016/j.bspc.2024.106023.
8. Bucholc M, James C. Khleifat AA, Badhwar A, Clarke N, Dehsarvi A, et al. (2023). "Artificial intelligence for dementia research methods optimization". Alzheimer's Dement. 19: 5934–51. doi:10.1002/alz.13441.
9. Chang CH. Lin CH, and Lane HY (2021). "Machine learning and novel biomarkers for the diagnosis of Alzheimer's disease". Int. J. Mol. Sci. 22:2761. doi:10.3390/ijms22052761.
10. Cheng F and Cummings J.(2022). "Artificial intelligence in Alzheimer's drug discovery". Alzheimer's disease drug development: Research and development ecosystem, eds Cummings J., Kinney J., Fillit H. (Cambridge: Cambridge University Press), 62–72. doi:10.1017/9781108975759.007.
11. Christodoulou RC, Woodward A, Pitsillos R, Ibrahim R, and Georgiou MF (2025). "Artificial intelligence in Alzheimer's disease diagnosis and prognosis using PET-MRI: A narrative review of high-impact literature post-Tauvid approval". J. Clin. Med. 14(16):5913. doi: 10.3390/jcm14165913.
12. Fabrizio C, Termine A, Caltagirone2 C, and

Sancesario G (2021). “Artificial intelligence for Alzheimer’s disease: Promise or Challenge”. Diagnostics (Basel) 11(8):1473. doi: 10.3390/diahgnostics110813473.

13. Fang J, Zhang P, Wang Q, Chiang CW, Zhou Y, Hou Y, et al. (2022). “Artificial intelligence framework identifies candidate targets for drug repurposing in Alzheimer’s disease”. Alzheimer’s Res. Therapy 14:7. doi:10.1186/s13195-021-00951-z.
14. Fang M, Hu J, Weiss J, Knopman DS, Albert M, Windham BG, Walker KA, Sharrett AR, Gottesman RF, Lutsey PL, Mosley T, Selvin E, and Coresh J (2025). “Lifetime risk and projected burden of dementia.” Nat Med 31(3),772-6.
15. Feng T (2023). “Applications of artificial intelligence to diagnosis of neurodegenerative diseases”. Stud. Health Tech. Inform. 308:648-55. doi: 10.3233/SHTI230896.
16. Fymat AL (2018a). “Alzheimer's Disease: A Review,” Journal of Current Opinions in Neurological Science 2(2);415-36.
17. Fymat, AL (2018b). “Alzheimer's Disease: Prevention, Delay, Minimization, and Reversal, Journal of Clinical Research in Neurology 1(1):1-16.
18. Fymat AL (2018c) “Is Alzheimer's an Autoimmune Disease Gone Rogue”, Journal of Clinical Research in Neurology 2(1):1-4, 2018.
19. Fymat, AL (2018d). “Is Alzheimer's a Runaway Autoimmune Disease? And How to Cure It? Proceedings of the European Union Academy of Sciences, 2018 Newsletter, pages 379-83.
20. Fymat AL (2018). “Dementia Treatment: Where Do We Stand“? Journal of Current Opinions in Neurological Science 3(1):1-3. 599.603.
21. Fymat AL (2019a). “Alzhei … Who? Demystifying the Disease and What You Can Do About it”, Tellwell Talent Publishers, pp 236. ISBN: 978-0-2288-2420-6 ; 978-0-2288-2419-0.
22. Fymat AL. (2019b). “On Dementia and Other Cognitive Disorders, Journal of Clinical Research in Neurology 2(1):1-14.
23. Fymat AL (2019c). “Dementia: A Review,” Journal of Clinical Psychiatry and Neuroscience 1(3):27-34.
24. Fymat AL (2019d). “Dementia with Lewy Bodies: A Review,” Journal of Current Opinions in Neurological Science 4(1);15-32.
25. Fymat AL (2019e). “What do we Know about Lewy Body Dementia”? Journal of Psychiatry and Psychotherapy (Invited Editorial) 2(1)-013:1-4. doi:10.31579/JPP.2019/018.
26. Fymat, AL (2020a). “Parkin... ss..oo..nn: Elucidating the Disease... and What You Can Do About it,” Tellwell Talent Publishers, pp 258. ISBN: 978-0-2288-2874-7; 978-0-2228-2875-4.
27. Fymat AL (2020b). “Dementia: Fending off the Menacing Disease... and What You Can Do About it”, Tellwell Talent Publishers, pp 488. ISBN: 978-0-2288-4146-3;978-0-2288-4145-6.
28. Fymat AL (2020c). “Alzheimer's: What do we Know about the Disease and What Can Be Done About It?” EC Journal of Psychology & Psychiatry 9(11):69-74.
29. Fymat AL (2020c). “Is Alzheimer's an Autoimmune Disease Gone Rogue? The Role of Brain Immunotherapy”, Journal of Clinical Research in Neurology 3(2):1-3.
30. Fymat, AL (2020d). “Alzheimer's: Will there ever be a Cure?” Journal of Clinical Psychiatry and Neuroscience 3(4): 1-5.
31. Fymat AL (2020e). “Dementia: Should we Reorient our Approach to Treatment”? EC Journal of Psychology & Psychiatry 9(12):1-3.
32. Fymat AL (2020f). “Dementia – What is its Causal Etiology?” International Journal of Neuropsychology and Behavioral Sciences

1(1):19-22.

33. Fymat AL (2021a). “On Potentially Reversible Forms of Dementia,” Journal of Current Opinions in Neurological Science 6(1):101-8.
34. Fymat AL (2021b). “Dementia – Eliminating its Potentially Reversible Forms,” Proc. European Union Academy of Sciences. Pages 270-7.
35. Fymat, AL (2022a). “Alzheimer's Disease: A Path to a Cure”, Journal of Neurology and Psychology Research 3(1):1-15. https://researchnovelty.com/articles.php?journal_id=5
36. Fymat AL (2022b). “Alzheimer's Disease: A Path to a Cure”, Current Opinions in Neurological Science 3(1):1-16.
37. Fymat AL (2024). “The Coming Dementia Pandemic: Prescription for a Cure,” Current Opinions in Neurological Science (Invited Editorial), 9(1):1-5.
38. Fymat AL (2025a). “Alzheimer’s Disease: Potential Application of Hyperbaric Oxygen Therapy”, Journal of Neurology and Psychology Research 5(6):1-20.
39. Fymat AL (2025b). “Prion-like Mechanisms in Neurodegenerative Diseases: I. Alzheimer’s Disease,” Journal of Neurology & Psychology Research 6(4):1-22.
40. Fymat AL (2025c). “A Novel Form of Dementia: Limbic-predominant Age-related TDP-43 Encephalopathy”, Journal of Neurology and Psychology Research 6(3):1-13.
41. Gu Z, Ge B, Wang Y, Gong Y, and Qi M (2025). “Artificial intelligence technologies for enhancing neurofunctionalities: A comprehensive review with applications in Alzheimer’s disease research”. Front. Aging Sci. 17:1609063. doi: 10.3389/fnagi.2025.1609063.
42. Hampel H. Vergallo A. Perry G.,and Lista S (2019). “Alzheimer Precision Medicine Initiative” J. Alzheimer’s Dis. 68:1–24. doi: 10.3233/JAD-181121
43. Harrer S. Shah P. Antony B. and Hu J (2019). “Artificial intelligence for clinical trial design”. Trends Pharmacol. Sci. 40:577–91. doi: 10.1016/j.tips.2019.05.005
44. Hemalatha B, Venkatachalam, Siuly S, and Cho J (2026). “AI driven framework for accurate detection of Alzheimer’s disease in EEG“. Nature Scientific Report 16: 5509.
45. Kale M, Wankhede N, Pawar R, Ballal S, Kumawat R, Goswami M, Khalid M, Taksande B, Upaganlawar A, Umekar M, Kopalli SR, and Koppula S (2024). “AI comprehensive review with applications in Alzheimer’s disease research”. Ageing Res Rev. 01:102497. doi: 10.1016/j.arr.2024.102497.
46. Li X, Feng X, Sun X, Hou N, Han F and Liu Y (2022). “Global, regional, and national burden of Alzheimer’s disease and other dementias, 1990–2019”. Front Aging Neurosci 14,937486.
47. Maturi MH, Satish S, Gonaygunta H, and Meduri K (2022). “The intersection of artificial intelligence and neuroscience: Unlocking the mysteries of the brain.” Int. J. Creat. Res. Comp. Technol. Design 4:1–21.
48. Maudsley S, Devanarayan V. Martin B. and Geerts H (2018). “Intelligent and effective informatic deconvolution of “Big Data” and its future impact on the quantitative nature of neurodegenerative disease therapy.” Alzheimer’s Dement. 14:961–75. doi:10.1016/j.jalz.2018.01.014.
49. Mayo Clinic (2025). “How is artificial intelligence helping to diagnose dementia?”
50. Mishra R and Li B (2020). “The application of artificial intelligence in the genetic study of Alzheimer’s disease”. Aging Dis. 11:1567–14. doi: 10.14336/AD.2020.0312.
51. (U.S.) National Institute oi Health (NIH) (2021). “The use of artificial intelligence in Alzheimer’s disease: Personalized diagnostics

and therapy".

52. Patel UK, Anwar A, Saleem S, Malik P, Rasul B, Patel K, et al. (2021). "Artificial intelligence as an emerging technology in the current care of neurological disorders." J. Neurol. 268:1623–42. doi:10.1007/s00415-019-09518-3.
53. Richards BA, Lillicrap TP. Beaudoin P. Bengio Y. Bogacz R. Christensen A. Clopath C, Costa RP. de Berker A. and Ganguli S (2019). "A deep learning framework for neuroscience". Nat. Neurosci. 22:1761–10.

doi: 10.1038/s41593-019-0520-2.

54. ScienceDirect.com (2021). "Application of artificial intelligence techniques for the detection of Alzheimer's disease using structural MRI images". Cybernetics & Biomedical Engineering 41(2):456-73.
55. Seo Y, Jang H, and Lee H (2022). "Potential applications of artificial intelligence in clinical trials for Alzheimer's disease". Life (Basel) 12(2):275. doi: 10.3390/life12020275.
56. Silva-Spínola A, Baldeiras I, Arrais JP, and Santana I (2022). "The road to personalized medicine in Alzheimer's disease: The use of artificial intelligence." Biomedicines 10:315. doi:10.3390/biomedicines10020315.
57. Subasi A, Kapadnis MN, and Bulbul AK (2022). "Alzheimer's disease detection using artificial intelligence" in Augmenting neurological disorder prediction and rehabilitation using artificial intelligence. Amsterdam: Elsevier.
58. Swargiary K (2024). Artificial Intelligence and Alzheimer's Disease, ERA, US. Washington, DC.
59. Valliani AAA, Ranti D, and Oermann EK. (2019). "Deep learning and neurology: A systematic review". Neurol. Ther. 8:351–65. doi: 10.1007/s40120-019-00153-8.
60. Wiltfang J, Esselmann H, and Barnikol UB (2021). "The use of artificial intelligence in Alzheimer's disease - Personalized diagnostics and therapy". Psychiatr. Prax. 48(S 01):S31-S36. doi: 10.1055/a-1369-3133. [Article in German].
61. Yao L, Ni J, Wei M, Li T, Li F, Wang T, Xiao W, Shi J, and Tian J (2025). "Recent advances in the application of artificial intelligence in Alzheimer's disease". Curr. Alzheimer Res. 22(11):815-27. doi:10.2174/0115672050410489250930175402.
62. Zhang M. Schmitt-Ulms G. Sato C. Xi Z, Zhang Y. Zhou Y, St George-Hyslop P.and Rogaeva E. (2016). "Drug repositioning for Alzheimer's disease based on systematic 'Omics'. Data Mining. 11:e0168812. doi: 10.1371/journal.pone.0168812.
63. Zhao X, Ang CKE, Acharya R, and Cheong KH (2021). "Application of artificial Iitelligence techniques for the detection of Alzheimer's disease using structural MRI images".Biocybernetics & Biomed. Eng. 41(2):456-73. https://doi.org/10.1016/j.bbe.2021.02.006