Advancements Build Momentum: 10 Years of Alzheimer’s Disease and Related Dementias Research
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Introduction

An estimated 6.7 million Americans are currently living with Alzheimer’s disease. Worldwide, more than 50 million people have dementia, a diagnosis that may include Alzheimer’s or a related disorder such as frontotemporal disorders, Lewy body dementia, or vascular dementias. This public health challenge takes a tremendous emotional, physical, and financial toll on those living with these diseases and their caregivers.

The National Plan to Address Alzheimer’s Disease, which arose from the National Alzheimer’s Project Act, spurred a substantial increase in federal funding for dementia research. As a result, over the past 10 years, the National Institutes of Health (NIH), led by its National Institute on Aging (NIA) and National Institute of Neurological Disorders and Stroke (NINDS), significantly expanded its investments in Alzheimer’s and related dementias research across the United States and beyond. Through enhanced collaboration and innovative partnerships with industry, other agencies, and people living with dementia and their families, NIH has:

» Advanced understanding of the risk factors, genetics, and mechanisms of disease in dementia
» Diversified and de-risked the therapeutic pipeline for disease-modifying drugs
» Advanced drug repurposing and combination therapy development
» Discovered tools to detect, diagnose, and monitor dementia
» Advanced clinical research on lifestyle interventions
» Increased understanding of how social and physical environmental factors affect dementia risk and disparities
» Expanded research on dementia care and care partner supports

Worldwide, more than 50 million people have dementia, a diagnosis that may include Alzheimer’s or a related disorder such as frontotemporal disorders, Lewy body dementia, or vascular dementias.

Over the past 10 years, the NIH, led by NIA and NINDS, significantly expanded its investments in Alzheimer’s and related dementias research.
A Decade of Progress

SPOTLIGHT

Translating Discoveries to the Clinic

The increased federal investment in Alzheimer’s and related dementias research in recent years yielded discoveries that advanced diagnostics, treatments, and potential preventions.

For example, the anti-amyloid antibody lecanemab-irmb (Leqembi) received FDA approval in 2023 based on a demonstrated effect in slowing cognitive decline. It is the first traditional (full) approval of a treatment that affects the underlying disease process of Alzheimer’s instead of only treating the symptoms of the disease. NIH support laid essential groundwork for the pharmaceutical company trials that led to this decision. Not only was the agency’s funding integral in understanding the role of amyloid, the protein targeted by lecanemab, the industry trials hinged on the use of amyloid PET imaging, a technology developed with NIH-funded research. Researchers also recently found that donanemab, another anti-amyloid drug, was effective in slowing the rates of cognitive and functional decline in participants who have early symptoms of Alzheimer’s.
Advanced Understanding of the Risk Factors, Genetics, and Mechanisms of Disease in Dementia

Ten years ago, scientists knew of only 10 genes linked to Alzheimer’s disease; today, we know of more than 70 relevant genetic regions, findings made in large part thanks to NIH funding. And although we knew years ago that the \textit{APOE} ε4 variant of the \textit{APOE} gene is a significant genetic risk factor for Alzheimer’s, we did not know why. Today, scientists know much more about the function of APOE protein — for example, it seems to influence all forms of dementia.
NIH-funded research suggests that the APOE ε4 gene can cause brain cells to build up abnormal amounts of lipids instead of using them to insulate nerve fibers. Another study found that high levels of APOE4 protein in neurons trigger an immune pathway that leads to tau tangles and cell death. These findings are leading to new approaches in developing potential dementia therapeutics.

Still, the genetics of Alzheimer’s is complex. NIH-funded researchers found that APOE ε4 is not as strong a predictor of risk in certain ethnic and racial groups, including those of African and American Indian ancestry, as it is in people of European ancestry. These findings underscore the importance of conducting more diverse population studies to determine specific genetic risk factors across racial and ethnic groups.

Beyond risk genes, scientists are also uncovering rare gene variants that may help protect against Alzheimer’s disease. These include an APOE variant called APOE3ch and a variant of the RELN gene called RELN-COLBOS. Understanding how these rare variants promote dementia resilience opens up new avenues for developing treatments.

**Decoding the genetics of Alzheimer’s-related dementias**

Scientists have also advanced our understanding of the genetic underpinnings of related dementias. For example, we now know that genetic cases of frontotemporal dementia are mostly caused by mutations in one of three genes: C9ORF72, MAPT, or GRN. A recent NIH-funded study revealed how specific mutations in these genes affect the age of onset and duration of frontotemporal dementias, which vary considerably. Understanding the causes of variation in age of onset could provide important clues about what causes frontotemporal dementias.

NIH scientists have also found two new genes involved in Lewy body dementia, demonstrating how risk genes often overlap across neurodegenerative diseases: One of these genes, BIN1, is also linked to Alzheimer’s disease, while the other, TMEM175, is involved in Parkinson’s disease.

**Identifying shared mechanisms of Alzheimer’s disease and mixed dementias**

As research continues, scientists are finding more commonalities across different forms of dementia. Once considered completely separate, brain changes found in Alzheimer’s, Lewy body dementias, frontotemporal dementias, and vascular dementias often overlap. This includes sharing risk factors, such as APOE ε4 and other genes, and associated disease processes, such as buildup and spread of misfolded proteins in the brain and loss of synaptic connections between neurons.

It is now understood that the pathologies thought to define distinct forms of dementia commonly co-occur: This is called mixed dementia. For example, Alzheimer’s pathology (beta-amyloid plaques and tau tangles) most commonly co-occurs with cerebrovascular disease (problems with blood vessels and blood flow in the brain) or Lewy bodies (alpha-synuclein clumps). Moreover, abnormal forms of a protein called TDP-43 are not limited to frontotemporal dementia but are commonly found with Alzheimer’s pathology and hippocampal sclerosis. Recently, another misfolded protein, TMEM106B, was also linked to diagnoses of Alzheimer’s, Lewy body dementia, and frontotemporal dementias.

Scientists continue to learn how having multiple dementias may shape the course of disease, paving the way for personalized treatment approaches.

**Discovering a common and under-recognized form of dementia**

Our appreciation of the complexities and forms of dementia continues to evolve. Research from the NIA Alzheimer’s Disease Research Centers recently led to the discovery and classification of a new form of dementia called limbic-predominant age-related TDP-43 encephalopathy (LATE). LATE is relatively common, and clinical symptoms associated with it mimic the clinical features of Alzheimer’s, meaning that some people clinically diagnosed with Alzheimer’s may have more LATE pathology than typical Alzheimer’s pathology.

Though it is not yet possible to identify LATE during life, researchers are investigating ways to diagnose it in living people. This would help in determining the most effective treatments, including personalized therapies, and in the selection of appropriate participants for dementia studies.
Investigating the higher risk of Alzheimer’s in women and other groups

Women have a higher risk than men of developing Alzheimer’s over their lifetimes. NIH-funded researchers are working to discover why. Recently, studies pointed to having an additional copy of certain genes, hormonal changes during menopause, and differences in how the brain makes energy as potential causes of the differences.

Additionally, people with Down syndrome are at high risk of developing Alzheimer’s because they carry an extra copy of the amyloid precursor protein gene. NIH-funded researchers found that Alzheimer’s progression in Down syndrome is similar to other genetic, early onset forms of the disease. This suggests that those living with Down syndrome may benefit from participating in studies — some of which are already in progress — on Alzheimer’s therapies aimed at slowing formation of amyloid plaques.

Looking beyond the brain to the gut and liver

Ten years ago, dementia research focused almost solely on the brain. Today, NIH-funded researchers are exploring other organ associations, including links between the digestive and vascular systems. For example, the gut microbiome’s role in inflammation and other disease processes has been associated with dementia in several studies. Ongoing NIH-funded microbiome research may also help explain why subgroups of people with Alzheimer’s respond differently to dietary and other interventions.

Research also points to a gut-liver-brain connection in dementia through the production of bile acids as well as key lipids that are needed by the brain.

Uncovering vascular contributions to cognitive impairment and dementia

Vascular dementias are no longer considered a separate form of dementia. More than 50% of dementia cases also show damage to the brain’s vascular system, which is made up of blood vessels that supply oxygen and other nutrients to the brain. Commonly identified through MRI scans, this damage is termed “diffuse white matter disease.” NIH-funded scientists are exploring this connection — known as vascular contributions to cognitive impairment and dementia — to better understand how and when vascular damage occurs. For example, their research has shown that key dementia proteins such as beta-amyloid can build up in the brain’s blood vessels or alter blood vessel formation and function. Injury to these affected blood vessels is a fairly common complication of the anti-amyloid therapies and can lead to brain swelling and bleeding in the brain.

Understanding neuropsychiatric symptoms in dementia

Neuropsychiatric symptoms such as psychosis, agitation, depression, sleep disturbance, and apathy are quite common in dementia. They can be among the most terrifying symptoms to patients, distressing to their care partners and families, and greatly affect quality of life. Among other initiatives to further study these symptoms, NIA collaborated with the NIH National Institute of Mental Health to launch the Psych-AD program, which is designed to better understand the molecular underpinnings of neuropsychiatric symptoms in dementia. The aim is to discover better biomarkers and targets for treatment of these symptoms.
Over the past decade, NIH funding has enabled the development and testing of 18 new dementia drug candidates in clinical trials, with two more ready to enter trials.

### NIA-Supported Drug Candidates That Have Advanced To Clinical Development

<table>
<thead>
<tr>
<th>Drug Candidate/Therapy Type</th>
<th>Targeted Biology (CADRO Theme)</th>
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<tbody>
<tr>
<td>AV-1959D (DNA vaccine)</td>
<td>Amyloid beta</td>
</tr>
<tr>
<td>AAV2-BDNF (Gene Therapy)</td>
<td>Growth Factors and Hormones</td>
</tr>
<tr>
<td>ACU193 (Immunotherapy - Monoclonal Antibody)</td>
<td>Amyloid beta</td>
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<tr>
<td>BMS-984923</td>
<td>Neurotransmitter Receptors</td>
</tr>
<tr>
<td>MW150</td>
<td>Inflammation</td>
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<tr>
<td>MW151</td>
<td>Inflammation</td>
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<tr>
<td>Posiphen</td>
<td>Proteostasis/Proteinopathies</td>
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<tr>
<td>OLX-07010</td>
<td>Tau</td>
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<tr>
<td>CS6253</td>
<td>ApoE, Lipids and Lipoprotein Receptors</td>
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<tr>
<td>NNI-362</td>
<td>Neurogenesis</td>
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<tr>
<td>J147</td>
<td>Metabolism and Bioenergetics</td>
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<tr>
<td>CMS121</td>
<td>Multi-target</td>
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<tr>
<td>Allopregnanolone</td>
<td>Multi-target</td>
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<tr>
<td>PU-AD/PU-HZ151/capamespib</td>
<td>Proteostasis/Proteinopathies</td>
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<tr>
<td>MW189</td>
<td>Inflammation</td>
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<tr>
<td>LM11A-31</td>
<td>Amyloid beta</td>
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<tr>
<td>CT1812</td>
<td>Amyloid beta</td>
</tr>
<tr>
<td>BPN14770/Zatolmilast</td>
<td>Neuroprotection/Resilience</td>
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Diversified and De-Risked the Therapeutic Pipeline for Disease-Modifying Drugs
Because of the substantial progress in understanding how dementia unfolds and NIH’s robust investment in drug development programs, the portfolio of drug candidates continues to expand and help de-risk further investment by the private sector. These drug candidates are intended to stop or slow the disease process rather than only treat symptoms, and some target amyloid plaques and tau tangles in new ways. Building upon our understanding of mechanisms of disease in dementia, most new drug candidates target other aspects of the disease, including problems with the immune system, proper protein assembly and removal, lipid balance, or metabolism.

**SPOTLIGHT**

**Diversity of drug targets in active clinical trials**

The breadth of mechanisms under study is increasing.

<table>
<thead>
<tr>
<th>Pharmacological</th>
<th>Phase I &amp; Phase II</th>
<th>Phase II/III &amp; Phase III</th>
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<tr>
<td>Targeted Disease Process</td>
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<td>Amyloid 4</td>
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<td>Inflammation 7</td>
<td>Synaptic Plasticity 1</td>
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<td>Receptors 7</td>
<td>Metabolism/Bioenergetics 1</td>
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<td>Neurogenesis 1</td>
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<td></td>
<td>Tau 1</td>
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**Total of 66 TRIALS**

**59 TRIALS**
Investing in Innovation for Tomorrow’s Breakthroughs

Through its open science approaches, NIH has accelerated the pace and efficiency of dementia research progress. Specifically, the agency's investments in centralized data-sharing platforms and other technologies make it possible for scientists to more freely share data, tissue samples, and other crucial research resources more broadly and effectively.

SPOTLIGHT

Anti-Amyloid Treatment in Asymptomatic Alzheimer’s Disease (A4) Study

The Anti-Amyloid Treatment in Asymptomatic Alzheimer’s Disease (A4) study, launched in 2014, is an ongoing prevention trial to test whether the drug solanezumab could slow cognitive decline associated with high brain amyloid if started before clinical symptoms of Alzheimer’s appeared. The findings were not positive, but they were definitive. Preliminary results showed solanezumab did not slow cognitive decline before clinical symptoms developed. However, information from the study has advanced our understanding of Alzheimer’s disease, and biosamples and detailed, patient-level data from the approximately 1,300 participants in the A4 trial will be available to the research community soon, enabling further discoveries about the disease and differences in the responsiveness to treatment.
Advanced Drug Repurposing and Combination Therapy Development

Drugs that are already FDA-approved to treat diseases other than dementia may hold a key to effectively treating Alzheimer’s and related disorders.
Researchers are exploring multiple ways to repurpose drugs either alone or in combination with another therapeutic through initiatives such as the NIA Advancing Combination Therapy and Drug Repurposing for Alzheimer’s Disease (ACTDRx AD) program. The Metformin in Alzheimer's Dementia Prevention (MAP) study, another late-stage trial in Phases 2b and 3, tests metformin, an FDA-approved medication for diabetes that has been repurposed to treat MCI and early-onset Alzheimer’s. MAP is testing the safety and effectiveness of metformin and is currently recruiting participants. This trial is projected to be completed in 2026.

**An antiepileptic drug to treat Alzheimer’s**

Thirty years of NIH-funded basic and translational research led to the discovery of a new mechanism for memory loss and the development of AGB101 as a possible new treatment for Alzheimer’s. AGB101 is a long-lasting version of the FDA-approved antiepileptic drug levetiracetam. Results from a recently completed Phase 3 clinical trial on the effectiveness of AGB101 for MCI caused by Alzheimer’s disease are forthcoming.

**A water pill for people at high genetic risk of Alzheimer’s**

By combining precision medicine and big data methods, an NIH-funded study identified bumetanide as a candidate for lowering the risk of Alzheimer’s disease in people who carry the APOE ε4 variant. Bumetanide is a common FDA-approved diuretic; further tests and clinical trials for its use in reducing Alzheimer’s risk are needed.

An NIH-funded study found a water pill may be a candidate for lowering the risk of Alzheimer’s disease in people who carry the APOE ε4 variant.
Discovered Tools to Detect, Diagnose, and Monitor Dementia

A timely and accurate dementia diagnosis is crucial for determining treatment options and selecting participants for the most relevant clinical trials. Before the early 2000s, autopsy was the only sure way to diagnose Alzheimer’s.

Currently, *biomarkers* are helping researchers diagnose dementia, monitor its progression, and gauge response to treatments. Often found in body fluids or with imaging, biomarkers are signs of what is happening in the body. Importantly, newer biomarkers can be collected non-invasively, which will make both clinical research and personalizing treatments for individual patients considerably easier.
Toward a biological definition of Alzheimer’s disease

NIH-funded research enabled the development of PET scans that detect abnormal beta-amyloid plaques in the brain, which can rule out other causes of cognitive impairment and support an Alzheimer’s diagnosis. More recently, PET scans have been developed to detect tau tangles, another hallmark of Alzheimer’s.

We can also measure proteins in an individual’s cerebrospinal fluid, sampled through a lumbar puncture, as a biomarker for abnormal beta-amyloid or tau in the brain. These imaging and spinal fluid biomarkers formed the basis of the 2018 NIA-AA Biological Research Framework, which has helped substantially in developing a biologically based definition of Alzheimer’s.

Blood-based biomarkers as a less expensive and less invasive option

An ideal biomarker test is minimally invasive and can be used in virtually any doctor’s office. Thanks in large part to NIH funding, the first blood-based biomarker test for Alzheimer’s is now available to many doctors, dependent on state-specific availability reflecting FDA guidelines, to help support diagnosis. Developed by C2N Diagnostics, the PrecivityAD™ blood test can accurately predict the presence of beta-amyloid plaques in the brain based on blood beta-amyloid levels, age, and APOE status.

The blood test is also being used in many clinical trials, such as to help select participants for the NIH-funded AHEAD Study. This trial is testing whether the newly FDA-approved drug lecanemab can prevent Alzheimer’s disease in at-risk people without symptoms. Scientists also continue to study different forms of the tau protein as a biomarker, including how well they work to predict dementia risk in different racial and ethnic groups. For example, in a multi-ethnic community-based sample, one form of tau was recently found to be a more accurate Alzheimer’s marker than beta-amyloid in diverse populations.

Emerging biomarkers

Discoveries in recent years point to new potential biomarkers, such as measuring brain inflammation or detecting abnormal TDP-43, which is found in frontotemporal dementias. Because of NIH funding, other biomarkers in development for detecting or monitoring dementia include the following:

- **Alpha-synuclein:** Clumped forms of alpha-synuclein are found in the brains of people with Parkinson’s disease and Lewy body dementias. A cerebrospinal fluid test for abnormal alpha-synuclein received FDA breakthrough device designation in 2019. Researchers are also developing a skin test for this biomarker that would be less invasive than a lumbar puncture.

- **Brain damage:** Researchers found that a protein called neurofilament light chain (NFL), which is released when nerve cells are damaged, may be a useful biomarker for familial Alzheimer’s and frontotemporal dementia.

- **Problems at the synapse:** Synapses are the spaces where neurons communicate with each other, but in Alzheimer’s and some other dementias, the synapses can stop working. In 2021, Alzheimer’s Disease Neuroimaging Initiative (ADNI) researchers identified a promising biomarker, NPTX2, for synaptic dysfunction and Alzheimer’s progression.

- **Vascular changes:** White matter lesions, which can be seen as bright white areas on brain images, have been linked with dementia. They have many causes, some of which are related to vascular changes, or changes in blood flow. A large, diverse cohort study is examining the role of white matter lesions in cognitive impairment and dementia.

Discover tools to detect, diagnose, and monitor dementia.
Discovering early indicators of dementia

In addition to these biomarkers, NIH-funded research has revealed other indicators that could help identify people in the earliest stages of dementia:

» **Money management:** People with dementia were more likely to miss credit card payments as early as six years before their diagnosis.

» **Driving behavior:** Several recent studies show how tracking driving behavior with GPS can help identify people with preclinical Alzheimer’s (who have biomarkers for the disease but do not have symptoms). These behaviors, such as hard braking, increased over time, corresponding with an increase in the biomarkers.

» **Early chronic pain:** People with dementia may experience increased levels of pain as early as 16 years before their dementia diagnosis. Though it is unlikely that pain causes or increases the risk for dementia, chronic pain may be an early indicator before other signs appear.

Digital Health Technologies Come of Age

NIH-funded researchers are advancing up-and-coming digital biomarkers for dementia, such as the use of digital devices to evaluate memory or learning through computer tasks. Recently, a tool that uses electronic health record data to detect unrecognized dementia was developed and validated.

For people diagnosed with dementia, wearable devices could enable doctors to monitor daily function and possible disease progression by tracking sleep patterns, gait, and mobility.
Advanced Clinical Research on Lifestyle Interventions

More scientific evidence is still needed, but strategies are emerging to potentially reduce dementia risk or delay its onset and progression. A 2017 evidence-based, NIA-commissioned report concluded that evidence on lifestyle factors to prevent Alzheimer’s, such as physical activity, blood pressure management, and cognitive training, is “encouraging although inconclusive.”

Since then, scientists have strengthened this knowledge base. For example, the NIH-funded SPRINT MIND trial indicates intensive blood pressure control may slow age-related brain damage and reduce the risk of MCI.
Brain training may improve some cognitive function

Past NIH-funded research has suggested that cognitive training improves certain aspects of cognitive function in older adults. A large, multisite clinical trial is currently assessing the efficacy of a similar cognitive training program to prevent or delay dementia onset. According to another NIH-funded study, engaging with new 3D virtual environments, such as 3D video games, may improve recognition memory.

Dietary and physical activity interventions

Dietary interventions continue to be a key focus of NIH research on dementia risk reduction. NIA-funded research found that in observational studies, the MIND diet, a hybrid Mediterranean-Dietary Approaches to Stop Hypertension (DASH) diet, was associated with a lower incidence of Alzheimer’s and a slower rate of cognitive decline. Other areas being investigated include multivitamins and a modified ketogenic diet.

Clinical trials are now in progress to test the effect of physical activity on cognitive function and dementia risk.

Emerging areas of focus

An emerging area of investigation is on interventions to enhance cognitive reserve, the mind’s ability to cope with the effects of aging. Other evolving focuses include interventions to potentially compensate for premature cognitive decline and dementia linked to adverse exposures in early life, such as neglect, abuse, and malnutrition.
Increased Understanding of How Social and Physical Environmental Factors Affect Dementia Risk and Disparities

Alzheimer's and related dementias do not affect all populations equally. Compared with White Americans, Hispanic Americans are 1.5 times as likely to develop dementia, and Black Americans are twice as likely. Despite this, NIH-funded research shows that dementia is under-diagnosed in these populations, pointing to the need for approaches to improve diagnoses in underserved communities.
Diving deeper into health disparities

Scientists are now uncovering biological mechanisms that underpin health disparities. Social stress, including discrimination, has been shown to contribute to accelerated aging of the immune system, which can play a key role in Alzheimer's disease. In addition, high blood pressure is a risk factor for dementia and is more prevalent in Black Americans than in other racial and ethnic groups in the United States.

Through ongoing clinical studies, researchers are testing culturally sensitive interventions aimed at reducing dementia disparities by addressing risk factors and improving the well-being of people with dementia. Other scientists are examining how to tackle larger-scale issues, including health care access and delivery.

Environment influences brain health

Many aspects of a person's life can affect their risk of developing dementia. These factors include everything from education and social status to where someone lives to their physical activity level. NIH-funded researchers are discovering how all these factors, collectively called the “exposome,” affect dementia.

For example, higher education levels may help preserve cognitive function and reduce the risk for dementia. Living near green spaces, such as parks and gardens, is also linked with higher cognitive function. In contrast, long-term exposure to air pollution raises the risk of dementia. Ongoing research is exploring how other aspects of the exposome, such workplace exposures and heavy metals, may contribute to dementia risk and disparities.

Population Studies Reveal Dementia Prevalence and Disparities

A recent NIH-funded study estimates that 10% of Americans age 65 and older have dementia and that 22% have MCI. Consistent with other studies, the researchers found higher prevalence for both Black and Hispanic Americans, as well as for people with lower levels of education.

Population Studies Uncover Dementia Risk and Protective Factors

NIH-funded research has uncovered new environmental, sociocultural, and behavioral factors throughout life that are associated with dementia. Understanding whether and how these factors affect dementia risk can point to potential prevention strategies to help lower that risk.

» Social engagement: Regularly seeing friends and family in midlife is linked to a lower risk of a dementia diagnosis later in life. In another study, women who were employed during early adulthood and midlife had slower rates of memory decline than those who did not work.

» Sleep: Insufficient sleep in middle age is associated with a higher risk of dementia later in life.

» Personality traits: Certain personality traits in adolescence are linked with lower dementia risk 50 years later. In another study, neuroticism (a tendency to feel self-doubt, anxiety, and other negative feelings) was linked with more amyloid and tau buildup in the brain, while conscientiousness was linked with less buildup.
Informed by its National Research Summit on Care, Services, and Supports for Persons Living with Dementia and Their Care Partners/Caregivers, NIH has significantly expanded research on how to improve dementia care and support for care partners over the past decade.
Identifying costs and challenges in dementia care

A 2021 NIH-funded study found that people with dementia who have an adult child available for caregiving are less likely to require paid care and transition into a nursing home. A growing movement to care for people with dementia outside of a nursing home setting means more hands-on care delivered in the community. Other studies are illuminating the costs of care, challenges, and need to support family care partners and have found the following:

» People with dementia and their families should shoulder more of the costs of care when these individuals live in the community, such as at home or with family members, than when they live in a nursing home or other residential facility. These costs do not factor in lost wages from caregiving.

» Paid care partners, such as home health aides, can help family caregivers to enable people with dementia to live safely at home and in the community. However, according to a 2020 study, only one in four community-dwelling individuals with dementia received paid care. People in the middle-income range were less likely than those with higher incomes or on Medicaid to receive paid care, underscoring the need to make paid care more accessible.

Improving quality of life for people with dementia and their care partners

Supporting care partners means assisting with the day-to-day challenges of caring for a loved one with dementia. In a 2021 evidence-based, NIH-commissioned report, the National Academies of Sciences, Engineering, and Medicine found two promising intervention types. These were collaborative care models that coordinate a dementia care team of experts to support care partners and REACH (Resources for Enhancing Alzheimer’s Caregiver Health) interventions that provide care partners with knowledge and resources.

Further dementia care intervention research will continue to expand the evidence base. In addition, telehealth programs developed and tested by NIH-funded researchers show promise in boosting dementia care and care partner support. Through these programs, people with dementia and their caregivers are provided with at-home access to care expertise and online care partner education.

Making an IMPACT

Launched in 2019, researchers in the IMbedded Pragmatic Alzheimer’s disease and related dementias Clinical Trials (IMPACT) Collaboratory test interventions to improve care of people with dementia in real-world settings. Projects currently underway include empowering emergency department nurses to improve detection of dementia in patients and strategies to improve dementia training and care management across interdisciplinary teams.

A 2021 IMPACT Collaboratory study found that COVID-19 was more severe in nursing home residents who were more cognitively impaired.
Looking Forward

Through sustained NIH investment, scientists have made significant strides in understanding Alzheimer’s and related dementias, and progress toward how to effectively diagnose, treat, and prevent them. Milestone therapeutic advancements mark the beginning of a new era of promise for the field and have reinforced the importance of pursuing amyloid as a strategic therapeutic target. These discoveries would not have been possible without the hard work and dedication of researchers, study participants, caregivers, and other stakeholders.

To ensure that dementia discoveries are broadly applicable, a top priority is for clinical trials and observational studies to better represent the diversity of the United States. Equally important is supporting a diverse dementia research workforce through Scientific Workforce Diversity Programs.

NIH is also expanding its dementia research portfolio into emerging scientific areas including precision environmental health. Researchers in this field study how factors in the environment, such as pollution, chemicals, and metals, interact with an individual’s genetics to affect the risk of dementia.

With continued commitment to scientific collaboration, data sharing, and innovative research, NIH is well positioned to fuel tomorrow’s breakthroughs.

Learn about future efforts in A New Era: Driving Momentum in Alzheimer’s and Related Dementias Research.
Appendix: References and Citations

Introduction


Section 1. Advanced Understanding of the Risk Factors, Genetics, and Mechanisms of Disease in Dementia


Section 3. Advanced Drug Repurposing and Combination Therapy Development


Section 4. Discovered Tools to Detect, Diagnose, and Monitor Dementia


Section 5. Advanced Clinical Research on Lifestyle Interventions


Section 6. Increased Understanding of How Social and Physical Environmental Factors Affect Dementia Risk and Disparities


