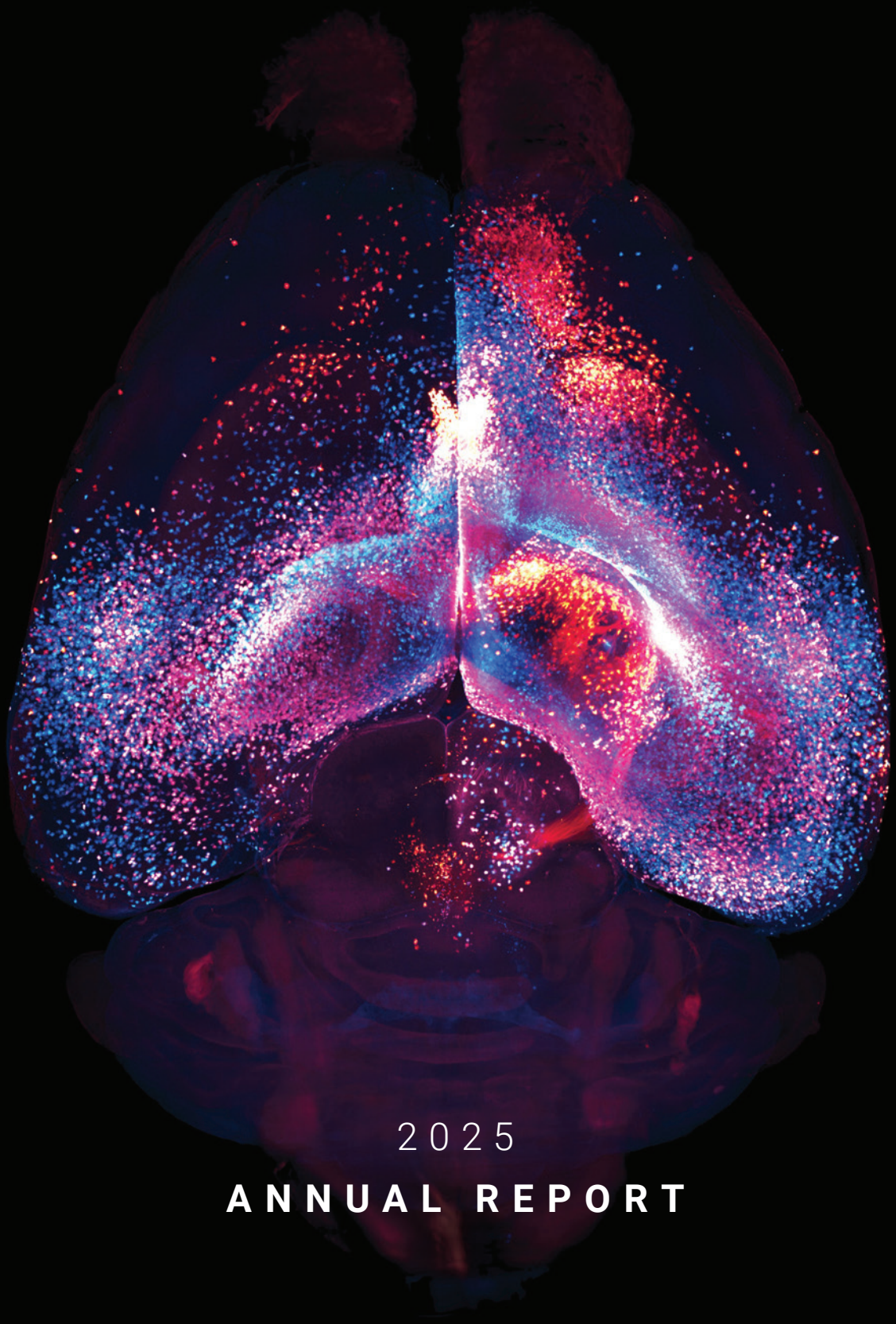
Cure Alzheimer's FUND



2025

ANNUAL REPORT

Resilience (noun):

The ability to withstand disruption, adapt to evolving conditions, and emerge stronger.

The year 2025 tested the resilience of our Cure Alzheimer's Fund community. Unprecedented disruptions in U.S. government funding and priorities challenged the biomedical research ecosystem that has yielded vital knowledge and progress in Alzheimer's and across human health. Yet our researchers and donors persevered, focused despite all distractions on our shared goal to end Alzheimer's disease. Together, we ensured that momentum was not lost and that progress toward our mission continued without pause.

OUR MISSION:

Cure Alzheimer's Fund is a nonprofit organization dedicated to funding research with the highest probability of preventing, slowing, or reversing Alzheimer's disease.

ON THE COVER:

"Astrocyte Galaxy." The brain relies on support cells called astrocytes, which form large, interconnected networks that work together to keep the brain functioning. These networks are not fixed—they can be built and remodeled based on experiences. For example, when an animal is deprived of certain sensations, specific networks shrink, showing that the brain's support infrastructure can adapt and reorganize. Understanding how these networks are maintained, disrupted, and reshaped helps reveal how the brain stays resilient in the face of challenges.

Three-dimensional, depth-coded light sheet imaging of a cleared adult mouse brain reveals vast, long-range astrocyte networks, labeled by a Cx43-TurboID astrocyte network tracer injected into the primary motor cortex. Each point marks an astrocyte coupled via connexin 43-mediated gap junctions, forming organized circuits that selectively link cortical and subcortical regions. The depth coding echoes astronomical images: astrocytes near the pial surface appear blue-shifted, while those extending toward deeper ventral regions shift to red, creating a "galaxy" of star-shaped glia across the brain.

The Liddelow lab recently discovered that these astrocyte networks are unexpectedly vast, connecting faraway brain regions and dynamically rewiring in response to experience and memory demands, revealing a new and powerful mechanism for brain coordination beyond neuronal circuitry.

IMAGE ATTRIBUTION: Melissa Cooper, Ph.D., is a postdoctoral researcher in the lab of Shane A. Liddelow, Ph.D., at New York University Grossman School of Medicine. She investigates astrocyte biology and neuroinflammation. Her work focuses on how astrocytes form networks across the forebrain and how reactive states of these astrocyte networks influence neural circuit function and disease progression, advancing potential therapeutic strategies for neurodegenerative and inflammatory brain disorders.

Report design: Winking Fish; Copy editor: Colleen O'Neill; Printer: Kirkwood

Dear Friends,

This year was unlike any we have seen before. When we first founded Cure Alzheimer's Fund, we knew there was a need to fund the high-risk, high-reward research that would seed the Alzheimer's research landscape with new ideas to help patients. With our funding, scientists have been able to pursue groundbreaking new hypotheses. With preliminary data developed thanks to CureAlz support, many went on to receive government funding—more than $649 million* in National Institutes of Health (NIH) funding resulted from our 2018–2022 grants—a necessary step to further move science from theory to therapy.

Never did we think this process would be upended the way it was in 2025. The federal government made repeated and unheralded sweeping changes to policies and funding that drastically impacted scientific research. Policies were announced and enforced with little notice; many were then revised or rescinded, all yielding significant instability in the scientific environment. The research community has been left reeling.

Partnering with the best scientific minds in Alzheimer's disease research has taught Cure Alzheimer's Fund that progress against this disease requires perseverance and adaptation in the face of obstacles, the very definition of resilience. Although the challenges of 2025 were unprecedented, we met them the same way we face every test: with laser focus on our mission. Our donors responded to our calls to action with record-breaking support, and we in turn immediately put that support to work, meeting the emergent needs of our researchers by growing our research program to $38.3 million, by far the largest amount in our history. As always, we shaped our support to optimize its impact, adding emergency funding and support for brain banks, and supporting laboratory staffing to stabilize the research pipeline. I am immensely proud and grateful for the resilience the CureAlz donor and researcher community enabled and demonstrated together in 2025. Despite all obstacles, we continue to propel science forward and get closer to a cure.

Henry F. McCance

As we finalized this annual report, I learned that my co-founder, Jacqui Morby, had passed away. Jacqui was an extraordinary woman who dedicated her life to pushing boundaries and improving the world around her. I am deeply saddened by this tremendous loss and can think of no greater tribute than continuing the work to find the cure for Alzheimer's disease she fought so passionately to achieve.

On behalf of myself, the Board of Directors, and Trustees, I sincerely thank everyone who contributed to this remarkable success.

Sincerely,

Henry F. McCance
Chair, Board of Directors

* For the combined years of 2018 through 2022, the total of $87 million in grants provided to researchers from Cure Alzheimer's Fund resulted in an extraordinary $649 million in follow-on National Institutes of Health/National Institute on Aging funding from 2018 through 2024.

Jacqueline ‘Jacqui’ Morby

1937–2026

“Our greatest goal is to be put out of business.”

This quote from Jacqui Morby encapsulates her drive to end the burden of Alzheimer’s disease. Despite being a Founder of Cure Alzheimer’s Fund and a Board member for more than 22 years, Jacqui wanted our organization to be purposeful but not permanent—the legacy she worked tirelessly for was the success of our mission.

Having lost her mother to the disease, she recognized that the best way to alleviate future suffering would be to accelerate the science vital to prevention, treatment, and cure. Jacqui never sought recognition for all she did for our mission, but instead always focused on the collaboration—among our scientists, with her fellow Founders and Board members, and among our CureAlz community—that made progress possible.

“Jacqui was central to shaping the original vision for Cure Alzheimer’s Fund,” said Tim Armour, former CEO and current CureAlz Board member. “The example she set of focusing on shared goals and knowledge rather than individual credit became part of our organizational ethos, and a requirement of the scientific collaborations CureAlz funds. Her quiet yet incisive leadership inspired each of us to do everything in our power to accelerate progress.”

Jacqui Morby was an extraordinary person whose warmth and humor never hinted at all that she had achieved professionally. She was a pioneer in the earliest days of venture capital, a rare woman in a field dominated by men, and one of the earliest investors to recognize the promise of software when the very term was little known.

Jacqui’s personal life was equally expansive. She and her husband, CureAlz Co-Founder Jeff Morby (1937–2023), embodied a partnership of true equals for 64 years, an inspiration to all who knew them. She was similarly devoted to her children, Drew and Michelle, and to her many friends. She and Jeff inspired many friends and colleagues to join them in their mission to end Alzheimer’s, and we are proud to count them in our CureAlz community.

We deeply mourn this loss and extend our condolences to all those who loved Jacqui. We are deeply grateful to Jacqui, and in tribute reaffirm our commitment to our mission.

Our success will be her legacy.

Cure Alzheimer's FUND

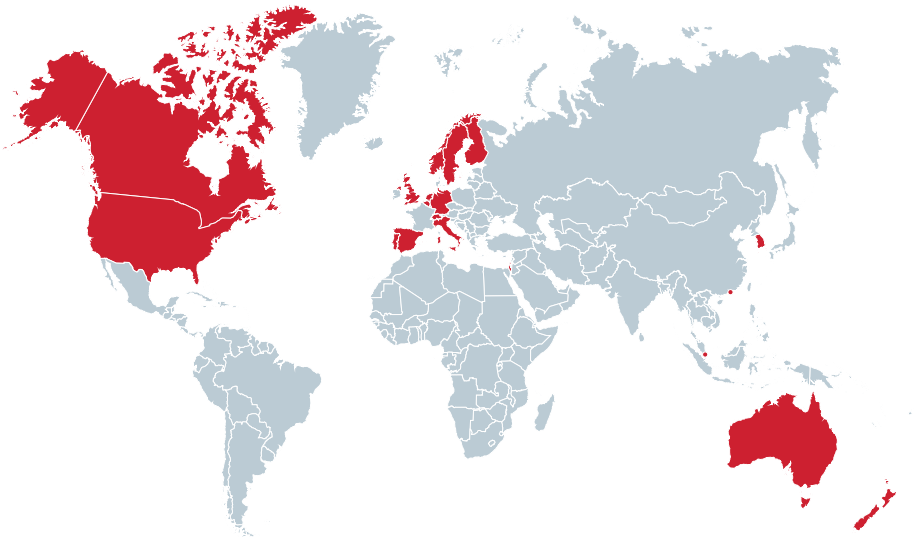
BY THE NUMBERS

Since our inception in 2004, we have focused on funding research with the most promising likelihood of breakthroughs. We're enabling the world's leading scientists to explore bold ideas and make game-changing discoveries.

384 FUNDED RESEARCHERS

131 FUNDED INSTITUTIONS

8 CONSORTIA



19 COUNTRIES WHERE CUREALZ FUNDS LABS:

- Australia
- Belgium
- Canada
- Finland
- Germany
- Hong Kong
- Israel
- Italy
- Netherlands
- New Zealand
- Norway
- Portugal
- Singapore
- South Korea
- Spain
- Sweden
- Switzerland
- United Kingdom
- United States

1,090 RESEARCH GRANTS

$261MM+ RESEARCH FUNDS DISTRIBUTED

1,823 RESEARCH PAPERS PUBLISHED

149,978 RESEARCH PAPER CITATIONS

You make this possible.

84,000+ DONORS

$341,000,000+ DONATIONS TO DRIVE RESEARCH FORWARD

All CureAlz figures above reflect inception in 2004 through Dec. 31, 2025.

A MESSAGE FROM OUR CHIEF EXECUTIVE OFFICER


Meg Smith

Our mission—pursuing a cure for a disease of the brain that strikes at the very core of who we are—is not for the faint-hearted.

Our progress has not been linear, and we know the path to success will be filled with further unexpected challenges and frustrating twists. Our mission is one of years, patience, determination, skill, and funding. But more than anything, it is a goal that will require a community to achieve, and I am truly grateful for ours.

Precipitous upheaval to government scientific funding and institutions massively destabilized American science in 2025. Researchers were left scrambling to ensure their research—and years of progress—were not lost. They appealed to Cure Alzheimer's Fund for a lifeline, and our donor community came to the rescue. It is because of you that I can report record numbers across the board for 2025, numbers that only begin to tell the story of how your support preserved vital science. We significantly surpassed all prior years in fundraising and in research distributions. To meet the needs of the moment, with the guidance of our Research Leadership Group, our committed staff deployed the additional resources you provided with incredible speed yet continued rigor and care. Notably, we provided unprecedented emergency funding to safeguard scientific progress and keep smart scientists in the lab—ensuring continuity today and discoveries tomorrow. Your commitment to our mission is inspiring.

The gratitude within the research community is fueling renewed momentum to advance science and bring more therapies to patients. Recent advances benefiting

2025 BY THE NUMBERS

LARGEST GIFT EVER

$47.2MM
total funds raised

$50MM
commitment over five years

11,783
donors

$38.3MM
research program spending

187
named researchers received funding

167
grants distributed

74
institutions received funding

early-stage patients—multiple diagnostic blood tests and two FDA-approved disease-modifying drugs—are testaments to the power of scientific perseverance and investment that inspire the field to push further and faster. The companies behind these drugs are investing in the next generation of this class of drugs and more patient-friendly administration methods. While we are encouraged to see this class of drugs becoming more accessible, these advances also push us to deepen our understanding so we can deliver preventive and meaningful treatments to everyone across the disease spectrum.

Thankfully, our ability to reduce our risk and support our brain health does not have to wait for pharmaceuticals. Drawing on epidemiological studies of hundreds of thousands of people, "The Lancet" Commission on dementia prevention estimates that 45% of dementia cases worldwide could be prevented or delayed by addressing modifiable risk factors, including some that we can affect as individuals. Evidence continues to mount that lifestyle interventions like high-fiber, well-balanced diets, regular exercise, and engagement with the people and world around us make a meaningful difference to our risk of developing dementia and Alzheimer's disease.

Cure Alzheimer's Fund continues to push research forward despite continued challenges, funding projects aimed at answering bold questions and connecting teams around the world to collaborate on promising science topics. This includes our Brain Aging Consortium that launched in 2025, and our consortium on sleep and circadian rhythms launching in 2026.

> "The gratitude within the research community is fueling renewed momentum to advance science and bring more therapies to patients."

Our Cure Alzheimer's Fund community makes all of this possible: our incredible Board of Directors, Trustees, and core group of donors who fund operations, our researchers, who are diligently searching for answers, our dedicated staff, and you, our donors, whose generosity fuels our scientific discoveries. Thank you to all who empower Cure Alzheimer's Fund in the fight against Alzheimer's and help us accelerate progress toward ending the burden of this disease.

Warmly,

Meg Smith
CEO

Transformational Gift Protects Vital Resources and Catalyzes Groundbreaking Discoveries

In 2025, Cure Alzheimer's Fund received the largest single contribution in our 20-year history of funding Alzheimer's disease research. This $50 million gift over five years will drive progress at a time when the United States government is downsizing and disrupting science in unprecedented ways. In 2025 alone, more than 7,500 grants were frozen or terminated, roughly 25,000 scientists and personnel left federal science agencies, and cuts and uncertainty forced academic institutions into hiring freezes, staff reductions, and scaled-back graduate training programs.

At this critical moment, The Cardinal Family Fund stepped up to preserve the work of early- and mid-career research scientists, critical investigations into precision medicine, and the research infrastructure and workforce needed for continued progress in Alzheimer's research. The fund addresses these challenges through three strategic initiatives.

The Cardinal Family Scholars Fund for Alzheimer's Disease Research

The Cardinal Family Scholars Fund for Alzheimer's Disease Research was established to provide stability for exceptional investigators during this critical period. The inaugural 2025 cohort of six investigators will pursue important research during this period of funding instability while developing the skills and recognition needed to become the field's next leaders.

The Cure Alzheimer's Fund Research Leadership Group selected these talented investigators for their scientific accomplishments, leadership potential, and active engagement in the Alzheimer's research community.

The Cardinal Family Scholars Fund 2025 inaugural class, from left: Hagen Tilgner, Ph.D.; Lulu Jiang, M.D., Ph.D.; Jorge Palop, Ph.D.; Andrew Yang, Ph.D.; Rachel Bennett, Ph.D.; and Doo Yeon Kim, Ph.D.

Each scholar receives supplemental support for an existing CureAlz-funded project, plus dedicated funds for training graduate students and postdocs, and flexible resources for laboratory essentials. Cure Alzheimer's Fund will amplify their work to raise their visibility. They will also receive training to develop critical science communication skills and practical messaging strategies to become effective ambassadors for Alzheimer's disease research. This combination of resources and training is critical to help early-career investigators compete for future funding, attract talented trainees, and establish themselves as emerging leaders.

The Cardinal Family Emergency Relief Fund

With this gift, Cure Alzheimer's Fund established The Cardinal Family Emergency Relief Fund to preserve momentum, protect skilled research teams, and ensure that promising Alzheimer's research continues despite federal funding shortfalls. In early 2025, projects approved and already underway suddenly faced funding interruptions and even cancellations, despite passing rigorous peer review and securing multiyear commitments. The disruption affected research at every stage: established teams with active experiments, graduate students and postdoctoral fellows pursuing their first independent studies, and even new projects that surpassed previously announced scoring standards for federal funding. This risked slowing scientific progress, dismantling research infrastructure, and driving talented investigators and trainees out of the field.

The fund operates through three components: The Cardinal Family General Emergency Fund, The Cardinal Family Emergency Trainee Fund, and The Cardinal Family High-Scoring Grants Fund. Together, these programs are a stabilizing force during a period of exceptional uncertainty in federal research funding.

The Cardinal Family Precision Medicine Research Fund

With the guidance of our external scientific leadership, Cure Alzheimer's Fund has always funded research seeking safe and effective prevention and therapeutics for every member of the Alzheimer's community. The Cardinal Family Precision Medicine Research Fund will advance research into how genetics, sex, ancestry, and environmental exposures interact to shape Alzheimer's risk and progression across populations, producing prevention strategies, diagnostics, and treatments that work for everyone.

In 2025, this funding also rescued brain banks facing closure or crippling cutbacks—these programs collect, preserve, and share generously donated human brain tissue—allowing scientists to study Alzheimer's directly in the organ it affects. This work validates biomarkers, reveals disease mechanisms, and ensures lab discoveries reflect what happens in humans. Without this intervention, decades of irreplaceable tissue samples, from an incredible array of patients and unaffected people, and the specialized expertise to maintain and share them with labs worldwide, would have been lost.

A Special Partnership for a Special Purpose

In 2025 CureAlz Board member and Rick Sharp Alzheimer's Foundation (RSAF) Chair Richard Birnbaum brought together three organizations united by a shared commitment to ending the burden of disease through vital scientific discovery and progress. Through this partnership, Cure Alzheimer's Fund, the Red Gates Foundation, and RSAF will support two key research initiatives over the next three years: the Brain Entry & Exit Consortium and the Sleep & Circadian Rhythms Consortium.

The Red Gates Foundation, established in 2020, is dedicated to empowering biomedical research to end the burden of human disease, while RSAF, founded in 2015, is dedicated to finding a cure for Alzheimer's. Together, these partners will contribute $6 million toward more than $10 million in total funding that Cure Alzheimer's Fund will deploy to support these cross-institutional, interdisciplinary consortia.

The Brain Entry & Exit Consortium investigates how age-related changes at the brain's borders influence disease risk and progression, while a new Sleep & Circadian Rhythms Consortium will assess how biological clock disruptions interact with Alzheimer's pathology.

This partnership strengthens our ability to move science forward—accelerating progress in critical areas of research and bringing us closer to meaningful advances in Alzheimer's prevention and treatment.

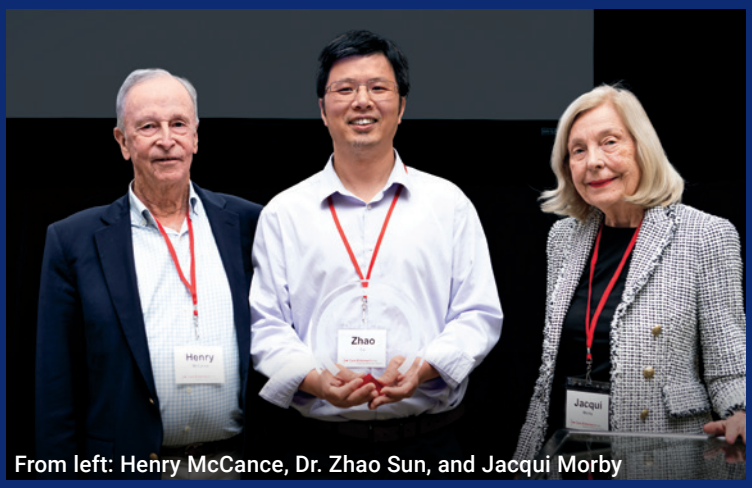
A New Way to Model Alzheimer's Earns 2025 Morby Prize

The second annual Jeffrey L. Morby Prize was awarded to senior author Andrew S. Yoo, Ph.D., and first author Zhao Sun, Ph.D., both of Washington University School of Medicine in St. Louis, for their paper, **"Modeling late-onset Alzheimer's disease neuropathology via direct neuronal reprogramming"** (Science, August 2024).

Their work introduces a new way to study neurons derived from patients while preserving key molecular and functional changes that develop over a lifetime. Past methods reset cells to an earlier "blank slate" state, erasing age-related characteristics and limiting their ability to model Alzheimer's disease. In contrast, this method reprograms patient skin cells directly into neurons, allowing researchers to retain important features of aging. These lab-grown neurons reflect the patient's own Alzheimer's-related pathology, including—for the first time in this type of model—amyloid beta plaques, tau tangles, and neurodegeneration. This advance offers a powerful new tool for studying the disease and testing potential therapies.

> "The generous support from the Jeffrey L. Morby Prize will further allow us to develop reprogramming approaches to generate different neuronal subtypes and investigate neuron-intrinsic aging mechanisms in the context of specific cell types."
>
> —DR. ANDREW S. YOO

Dr. Andrew S. Yoo



From left: Henry McCance, Dr. Zhao Sun, and Jacqui Morby

ABOUT THE JEFFREY L. MORBY PRIZE

In 2023, the Alzheimer's research community lost a visionary leader and Co-Founder of Cure Alzheimer's Fund, Jeffrey L. Morby. To honor his legacy, Cure Alzheimer's Fund established the annual Jeffrey L. Morby Prize. This prize recognizes the senior and first authors of a recent scientific publication that advances understanding of Alzheimer's disease and opens new paths toward prevention, diagnosis, or treatment. Recipients are selected by Cure Alzheimer's Fund–supported researchers and receive an award that includes funding to support ongoing Alzheimer's research in the senior author's lab. Honoring scientists whose work reflects innovation, rigor, and meaningful impact is a fitting tribute to Jeff's enduring commitment to advancing Alzheimer's research.

A Global Network to Advance Science

Collaboration and the open exchange of ideas define Cure Alzheimer's Fund's approach, creating a dynamic network among the researchers we support. By working across disciplines and institutions, our scientists build on one another's discoveries and accelerate the pace of Alzheimer's progress. These partnerships deepen our understanding of the disease and move us closer to meaningful, life-changing solutions.

Consortium Model

Cure Alzheimer's Fund supports both high-risk, high-potential individual grants and larger, multi-institution teams focused on key scientific areas. Our consortium model brings researchers from around the world together to collaborate, share insights, and exchange data—accelerating progress in critical areas of research.

▶ Alzheimer's Disease Tau Consortium

Tau tangles are a hallmark of Alzheimer's disease and closely track with the onset and progression of cognitive symptoms. This consortium focuses on understanding how tau pathology develops, how it is influenced by amyloid beta, and how it drives neurodegeneration—critical steps toward identifying effective interventions.

▶ Brain Entry & Exit Consortium

The brain depends on tightly regulated systems to allow nutrients in and remove waste. These protective borders and clearance pathways can become compromised with age and disease. This consortium investigates how these systems function—and how strengthening them could help prevent or slow Alzheimer's progression.

▶ Microbiome Consortium

The gut microbiome—home to trillions of microorganisms—plays an important role in overall health, including brain function. Emerging research suggests that changes in the microbiome may influence Alzheimer's risk and progression. This consortium seeks to better understand these connections and identify potential therapeutic targets.

▶ Brain Aging Consortium

(launched in 2025)

Aging is the greatest risk factor for Alzheimer's, and its effects vary widely from person to person. This consortium studies how aging contributes to disease risk, why some individuals develop Alzheimer's earlier, and why others remain cognitively resilient despite brain pathology. Supporting healthy brain aging is a vital strategy to reduce Alzheimer's.

▶ Fleming APOE Consortium

The APOE gene is one of the strongest genetic factors influencing Alzheimer's risk, and it plays a role in many biological pathways. Different variants increase risk (APOE4) or offer protection (APOE2). This consortium explores how APOE affects brain and body biology, with the goal of identifying new opportunities to intervene across multiple stages of the disease.

▶ Neuroimmune Consortium

The immune system plays a critical role in maintaining brain health, responding to threats and clearing debris. However, when this response becomes dysregulated, it can be damaging rather than beneficial. This consortium examines how the immune system interacts with the brain over time and how it can be directed to protect against Alzheimer's disease.

Learn from the Experts

Our webinar series shares the advances our community enables and raises awareness. In 2025, we hosted six webinars and recorded interviews with 10 researchers. Watch now: curealz.org/webinars.

Webinar speakers from left: Meg Smith, J.D.; Daniel Bos, M.D., Ph.D.; Karen E. Duff, Ph.D.; "Sunset and the Mockingbird" panel; Patricia Annino, J.D.; and Rudolph E. Tanzi, Ph.D.

Researcher Interviews included: Yun Chen, Ph.D.; Wade Self, Ph.D.; Piyali Saha, Ph.D.; Riqiang Yan, Ph.D.; Charles Glabe, Ph.D.; Shane A. Liddelow, Ph.D.; Laura M. Cox, Ph.D.; William C. Mobley, M.D., Ph.D.; Rudolph E. Tanzi, Ph.D.; and Ronald C. Petersen, M.D., Ph.D.

Scientific Leadership

Our scientific leaders work collaboratively to determine the highest scientific priorities for our research funding and to identify the research proposals with the highest potential to accelerate progress. Two advisory groups made up of esteemed researchers share their expertise, participate in several meetings throughout the year, and work closely with our staff to facilitate collaboration and disseminate research findings to the broader community.

RESEARCH LEADERSHIP GROUP (RLG)

The RLG includes 41 leading scientists in Alzheimer's and related fields. These leaders are the primary decision makers regarding our overall direction, as well as for specific proposals and projects. The RLG recruits investigators, conducts peer reviews on research proposals and reports, participates in quarterly meetings, and drives collaboration.

Randall J. Bateman, M.D.
Washington University School of Medicine in St. Louis

Mathew Blurton-Jones, Ph.D.
University of California, Irvine

Guojun Bu, Ph.D.
The Hong Kong University of Science and Technology

Oleg Butovsky, Ph.D.
Brigham and Women's Hospital; Harvard Medical School

Marco Colonna, M.D.
Washington University School of Medicine in St. Louis

Laura M. Cox, Ph.D.
Brigham and Women's Hospital; Harvard Medical School

Bart De Strooper, M.D., Ph.D.
VIB-KU Leuven, Belgium; University College London, England

Marc I. Diamond, M.D.
University of Texas Southwestern Medical Center

P. Murali Doraiswamy, MBBS, FRCP
Duke University School of Medicine

Karen E. Duff, Ph.D.
University College London, England

Caleb E. Finch, Ph.D.
University of Southern California

Li Gan, Ph.D.
Weill Cornell Medicine

Samuel E. Gandy, M.D., Ph.D.
Icahn School of Medicine at Mount Sinai

Charles Glabe, Ph.D.
University of California, Irvine

Alison M. Goate, D.Phil.
Icahn School of Medicine at Mount Sinai

Teresa Gomez-Isla, M.D.
Massachusetts General Hospital; Harvard Medical School

Christian Haass, Ph.D.
German Center for Neurodegenerative Diseases (DZNE), Germany

David M. Holtzman, M.D.
Washington University School of Medicine in St. Louis

Bradley T. Hyman, M.D., Ph.D.
Massachusetts General Hospital; Harvard Medical School

Costantino Iadecola, M.D.
Weill Cornell Medical College

Nancy Ip, Ph.D.
The Hong Kong University of Science and Technology

Jonathan Kipnis, Ph.D.
Washington University School of Medicine in St. Louis

Bruce Lamb, Ph.D.
Indiana University School of Medicine

Christoph Lange, Ph.D.
Harvard T.H. Chan School of Public Health

Cynthia A. Lemere, Ph.D.
Brigham and Women's Hospital; Harvard Medical School

Yueming Li, Ph.D.
Memorial Sloan Kettering Cancer Center

Shane A. Liddelow, Ph.D.
New York University

William C. Mobley, M.D., Ph.D.
University of California, San Diego

Ronald C. Petersen, M.D., Ph.D.
Mayo Clinic, Rochester

Leonard Petrucelli, Ph.D.
Mayo Clinic, Jacksonville

Sangram S. Sisodia, Ph.D.
The University of Chicago

Beth Stevens, Ph.D.
Boston Children's Hospital; Harvard Medical School; Broad Institute

Rudolph E. Tanzi, Ph.D. *Chair*
Massachusetts General Hospital; Harvard Medical School

Li-Huei Tsai, Ph.D.
Massachusetts Institute of Technology; Broad Institute

Robert Vassar, Ph.D.
Northwestern University Feinberg School of Medicine

Cheryl Wellington, Ph.D.
University of British Columbia, Canada

Stephen T.C. Wong, Ph.D.
Houston Methodist Research Institute; Weill Cornell Medicine

Tony Wyss-Coray, Ph.D.
Stanford University

Riqiang Yan, Ph.D.
University of Connecticut Health Center

Andrew S. Yoo, Ph.D.
Washington University School of Medicine in St. Louis

Hui Zheng, Ph.D.
Baylor College of Medicine

SCIENTIFIC ADVISORY BOARD (SAB)

The SAB provides guidance to Cure Alzheimer's Fund regarding its overall scientific direction and funding efficacy. The members—who have broad experience bringing therapeutics to patients—review the entire research portfolio to ensure that CureAlz is supporting investigations into the most important issues in Alzheimer's disease, and that our funding mechanisms accelerate the path to patients.

Vince Groppi, Ph.D.
Oricula Therapeutics

John S. Lazo, Ph.D.
University of Virginia

Patrick C. May, Ph.D.
ADvantage Neuroscience Consulting LLC

Steven M. Paul, M.D. *Chair*
Seaport Therapeutics

Finding the Resilience Within for a Better Future

We often define resilience as an aspect of our character, something we draw on when life is hard. But science continues to reveal new wonders of the brain, including that it can have its own form of resilience. For Alzheimer's disease, recognizing this resilience addresses one of the disease's longtime mysteries.

Not everyone who develops amyloid plaques and tau tangles goes on to develop cognitive decline and dementia. Doctors and scientists first discovered how brain function and brain pathology may not align when examining donated brains after patients died. As technology advanced and brain scans made it possible to detect plaques and tangles in living patients, researchers found that some older adults had brains laden with enough Alzheimer's pathology for a biological diagnosis, yet they were cognitively unimpaired. By understanding what makes these brains resilient to pathology, scientists realized they might be able to harness that power to help others.

Scientists are learning that not only do some brains naturally withstand damage, but all brains may be trained to become more resilient. In 2025, CureAlz funded research on both fronts and took unprecedented action to protect something equally important: the researchers doing the work.

Preserving Synapses to Protect Cognition

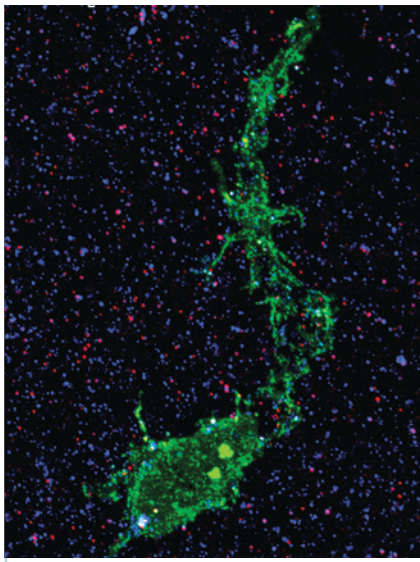
Alzheimer's disease is associated with amyloid plaques, tau tangles, and brain cell death, but science is revealing that synapse loss is equally important. In fact, synapse loss goes hand in hand with symptoms of the disease. It is also one of the best predictors of dementia severity.

Synapses are the communication points between neurons. Every thought, memory, perception, and behavior is the product of synaptic activity across billions of neurons. Synapse health is a measure of brain health.

During disease progression, synapse loss follows a specific path through the brain, and not all synapses are affected. For Alzheimer's, synapses in brain regions important for memory are affected, while synapses in areas like those controlling movement are preserved until very late in the disease, even though plaques and tangles are present.

This selective preservation—where some synapses are lost while others survive—is known as local resilience. Even more striking, some individuals display global resilience; their brains are full of plaques and tangles, yet they have no synapse loss or dementia. Together, these phenomena reveal that the brain has a remarkable capacity to resist the damage within it.

continued >



Microglial engulfment of synapses in a human Alzheimer's disease brain. A microglia, the brain's immune cell, in green, is shown digesting red and blue synapses, the communication points between brain cells.

Image Attribution: Sunny Kumar, Ph.D., and Charles Zachary Klein, B.S., from the lab of Teresa Gomez-Isla, M.D., at Mass General Brigham.

This raises a fascinating question: What is the difference between a vulnerable synapse and a resilient one, and can we shift the balance?

In 2025, Cure Alzheimer's Fund funded research studying why synapses are lost and how to protect them.

By studying what makes synapses resilient, scientists can learn how to protect vulnerable ones, so that even if plaques and tangles develop, scientists might still be able to protect what matters most—the connections that preserve memories and thoughts.

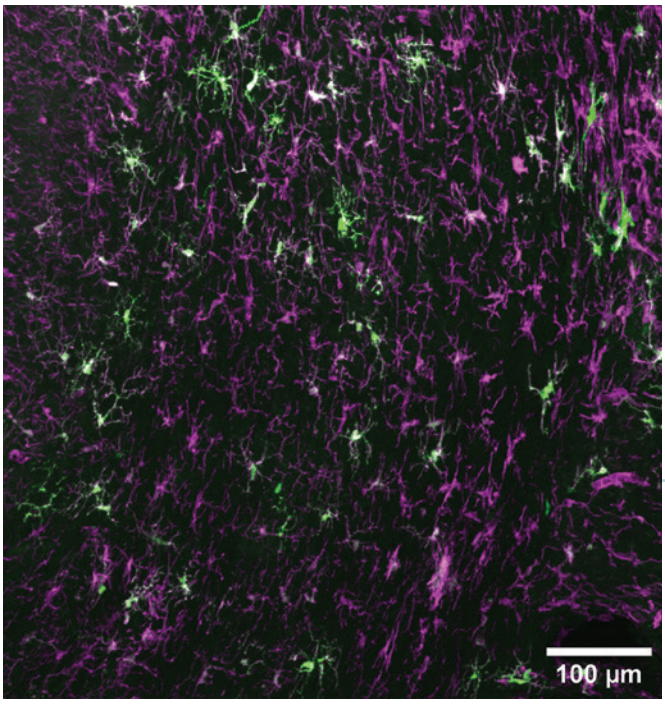
The Immune System and the Brain: A Lifelong Relationship

Our immune system has two parts. One is housed within the brain and dedicated to defending it. The other, known as the peripheral immune system, circulates through the blood to protect the rest of the body. For decades, scientists thought the two systems never interacted under healthy conditions because of the protective borders separating the brain from the rest of the body. But now we understand that rather than being impenetrable walls, these borders are sophisticated checkpoints that pass messages back and forth between the brain and the body's immune system. This allows the brain to monitor the body's immune status and the immune system to support and fine-tune brain function.

This checkpoint system may also help explain recent findings that certain vaccines, such as the shingles vaccine, can reduce the risk of Alzheimer's. This suggests that training the peripheral immune system—even against unrelated diseases—may somehow protect the brain. Scientists are only beginning to understand why, but it demonstrates that the two immune systems interact in ways that matter deeply for brain health and resilience to disease.

In 2025, Cure Alzheimer's Fund funded research into how the two immune systems communicate and what happens when this communication breaks down in ways that contribute to Alzheimer's disease.

Cure Alzheimer's Fund also funded research exploring whether and how vaccines build resilience against Alzheimer's. The work focused on a tuberculosis vaccine (BCG), which is known to have immune effects that extend beyond protection against tuberculosis. This research examined how the vaccine trains not only peripheral immune cells but also brain cells. The results were promising enough that this work has now advanced to a clinical trial to determine whether BCG can be used as protection against Alzheimer's.



Microglia in a mouse brain. Researchers successfully delivered a genetic marker (green) into microglia (purple), the brain's immune cells. This is the first step in using this same method to dial down the inflammation that drives Alzheimer's.

Image attribution: Paula Espinoza, B.S., from the lab of Casey A. Maguire, Ph.D., at Massachusetts General Hospital.

Supporting Scientific Resilience

Scientific advancement relies on a continuous pipeline of scientists at every stage of development—from graduate students and postdoctoral fellows to early- and mid-career researchers—to carry the work forward. A break anywhere in this pipeline can set the field back for decades.

In 2025, federal funding cuts for scientific and medical research threatened the integrity of this pipeline at multiple points.

Graduate and postdoctoral students form the backbone of active laboratories, receiving federal funding for their studies in return for lab work and classroom teaching. In 2025, these grant awards dropped to their lowest point in almost 10 years. Some students had their funding canceled with only months left to go in their programs. Without funding, they had to choose between leaving science or working without pay toward their credential.

Early- and mid-career scientists were disproportionately impacted by federal funding disruption. They depend on federal grants to build a lab, hire trainees, and pursue research. When federal funding disappeared, they faced an impossible choice: abandon their scientific work or struggle without the resources their labs need to function.

Cure Alzheimer's Fund recognized not only the immediate harm to active science projects, but also the long-term damage of losing an entire generation of Alzheimer's researchers. With donor support, Cure Alzheimer's Fund provided, for the first time in our history, short-term support to 21 researchers across career stages. By keeping scientific talent engaged in Alzheimer's research at a moment when many would have been forced out, Cure Alzheimer's Fund supported scientific resilience, protected the scientific pipeline, and ensured funded projects stayed on track.

Meaningful discoveries emerge through sustained effort, precision, and perseverance. Scientists analyze blood-based biomarkers linked to Alzheimer's disease. Each step contributes to improving how these markers are understood and eventually applied in clinical settings.

Image attribution: Isabella DiBerardino (front) and Zoe Horlick, pictured below, from the lab of Cheryl Wellington, Ph.D., at the Djavad Mowafaghian Centre for Brain Health, University of British Columbia.

Looking Forward

Resilience defined our community—donors and scientists alike—in our response to 2025, and as we look ahead to 2026 and beyond, we know it will remain essential to sustaining our work and advancing science. It is also proving to be a compelling lens through which to understand Alzheimer's disease.

New discoveries are teaching us that we can build resistance and resilience to Alzheimer's. By identifying the lifestyle factors and environmental exposures that protect the brain, or leave it vulnerable, we have the opportunity to translate science into real, actionable protection for people long before disease takes hold.

We will always fund research pursuing the pathways and targets needed to stop or slow Alzheimer's in those who need it, but we are equally committed to ensuring fewer people ever reach that point.

Building brain resilience requires understanding the factors that protect the brain, which is why, in 2026, we are launching the Sleep & Circadian Rhythms Consortium.

Formation of the Sleep & Circadian Rhythms Consortium

Sleep is something we all do, and science is revealing just how much it matters for brain health and Alzheimer's risk. The circadian rhythms that regulate our bodies' daily cycles are proving equally important. In fact, it is the circadian system that drives sleep itself, moving us through the cycles we need each night. When either is disrupted, the consequences for the brain can be significant.

Circadian rhythms coordinate timing across the entire body, and when they get out of sync, it can throw the body out of balance and create conditions that favor Alzheimer's pathology. Sleep shapes brain health and is essential for encoding memories and for clearing waste products produced during waking hours, such as toxic amyloid beta and tau. Science has shown that disruptions in sleep and circadian rhythms are linked to increased dementia risk, and that they seem to be both contributors to and consequences of emerging Alzheimer's pathology.

Scientists think sleep and circadian rhythms can be supported with lifestyle changes and other interventions, which may offer new ways to reduce Alzheimer's risk. But despite the growing evidence linking sleep, circadian rhythms, and Alzheimer's disease, we don't fully understand how they interact. That's why, in 2026, Cure Alzheimer's Fund is investing in this promising area by convening the Sleep & Circadian Rhythms Consortium. Composed of researchers from multiple disciplines, the consortium will work to understand how sleep and circadian rhythms influence Alzheimer's, and how that knowledge can be turned into real interventions.

Research Stages

In 2025, Cure Alzheimer's Fund achieved a record high in research investment, deploying **$38.3 million**—a 27% increase above 2024. Our program supported **167 grants** and **187 investigators** advancing work across the full spectrum of Alzheimer's research.

Foundational
$11,139,749
(29%)

Foundational research drives the earliest discoveries about Alzheimer's—where important questions remain unanswered and new insights can reshape the field. This work lays the foundation for future breakthroughs and the promise of more precise approaches to Alzheimer's prevention and treatment.

In 2025, funding supported research that reflects the diversity of people affected by Alzheimer's, including historically understudied groups. Projects included funding brain banks to ensure donated tissue reaches labs worldwide despite cuts in federal support, advancing the Alzheimer's Genome Project™ to better understand genetic risk and age of onset, and developing blood-based biomarkers to detect the disease earlier—before symptoms appear. We also launched the Brain Aging Consortium.

Translational
$24,025,338
(63%)

Translational research builds on foundational discoveries to move promising ideas closer to real-world impact. This work connects laboratory findings to approaches that ultimately may benefit patients.

In 2025, Cure Alzheimer's Fund supported research that explored how key proteins—such as amyloid beta, tau, and APOE—influence disease progression; how immune cells in the brain, like microglia and astrocytes, may either contribute to or protect against damage; how studying Down syndrome offers insights into amyloid beta Alzheimer's pathology; and more. This funding supported the Neuroimmune Consortium, the Fleming APOE Consortium, and the Brain Entry & Exit Consortium.

Drug Discovery and Enabling Technologies
$804,888
(2%)

Drug discovery research focuses on developing new approaches to prevent or treat Alzheimer's disease. In 2025, funding addressed one of the field's most persistent challenges: delivering treatments effectively to the brain. Researchers worked to engineer new gene therapy delivery systems that can cross the blood-brain barrier, a critical step in making advanced therapies viable.

We also supported the screening of potential drug candidates, including compounds designed to reduce toxic buildup and improve the brain's natural clearance processes. While these efforts are still in early development, they represent important steps toward treatments that do not yet exist.

Preclinical and Clinical Drug Development and Trials
$1,482,264
(4%)

At the preclinical and clinical stages, promising therapies are refined and tested to prepare them for use in human trials and ensure that the most effective and safest approaches can move forward.

In 2025, we supported scientists exploring new ways to target neuroinflammation, including therapies engineered to reach the brain without invasive procedures. Other efforts aimed to protect synapses—the connections between neurons that are essential for memory and cognition—using precision tools to reduce harmful tau activity. We also are supporting the development of inhibitors that can modify the epigenetic code and help restore the expression of healthy genes in affected brain cells.

Basic Research: Early Stage → Drug Development Process

CureAlz deployed 2% to support research operations, research materials, and scientific meetings.

2025 Funded Research

Prioritizing Returns Measured in Scientific Progress—Not Potential Profits

Cure Alzheimer's Fund measures success in scientific progress—not financial returns. In 2025, the majority of funding—92% of awards—supported early-stage research, prioritizing the identification of promising targets over proprietary drug development. The ideas we fund help advance the broader field, catalyzing new therapeutics and, when warranted, new companies. As discoveries progress, we continue to support the most promising work as it moves toward clinical application and, ultimately, patients.

Diversifying Our Research Portfolio by Following the Science

Each year, Cure Alzheimer's Fund strategically balances our research portfolio, extending vital stability to projects demonstrating compelling progress and creating opportunity for the most promising emerging ideas.

53% new projects

32% new investigators

15% new institutions

For more information about the 2025 research projects listed below, visit **curealz.org/2025research**.

Foundational Research

GENETIC RISK FACTORS

Mapping the X Chromosome Multi-Ome in Alzheimer's and Parkinson's Disease

Michael Belloy, Ph.D.
Washington University School of Medicine in St. Louis

Systematic Assessment of Tandem Repeats in Alzheimer's Disease (STaR-AD)

Lars Bertram, M.D.
Valerija Dobricic, Ph.D.
University of Lübeck, Germany

Integrating Single-Cell Genomics for Pathways to Protection and Resilience Against Alzheimer's Disease

Winston Hide, Ph.D.
Beth Israel Deaconess Medical Center; Harvard Medical School

Moving the Cure Alzheimer's Fund Alzheimer's Genome Project™ Beyond Simple Associations: Integrating Functional Information, Fine-Mapping and Causal Inference Approaches into the Family-Based Analysis of the Cure Alzheimer's Fund Whole-Genome Sequencing (WGS) Family Study

Christoph Lange, Ph.D.
Harvard T.H. Chan School of Public Health

Precision Medicine Prediction Model for Alzheimer's Disease Using Cooperative Learning Approaches for Multi-Omic Data

Christoph Lange, Ph.D.
Harvard T.H. Chan School of Public Health

Interpreting Alzheimer's Disease-Associated Genetic Variation at Enhancer Regions

Andreas R. Pfenning, Ph.D.
Carnegie Mellon University

The Alzheimer's Genome Project™

Rudolph E. Tanzi, Ph.D.
Massachusetts General Hospital; Harvard Medical School

BIOMARKERS, DIAGNOSTICS, AND STUDIES OF RISK AND RESILIENCE

BRAIN AGING CONSORTIUM:

Identifying Age-Related Proteomic Changes That Predict Future Onset of Amyloid-Beta Aggregation in Alzheimer's Disease

Randall J. Bateman, M.D.
Washington University School of Medicine in St. Louis

BRAIN AGING CONSORTIUM:

The 100-Plus Study Brain Cohort: Identifying Molecular Determinants of Resistance, Resilience and Early Cognitive Decline in the Oldest-Old

Henne Holstege, Ph.D.
VIB-KU Leuven Center for Neuroscience, Belgium

BRAIN AGING CONSORTIUM:

Brain Region and Cell-Type Specific Aging from Accelerated to Resilient Trajectories

Miranda E. Orr, Ph.D.
Washington University School of Medicine in St. Louis

BRAIN AGING CONSORTIUM:

Investigating Lipidomic Perturbations in the CSF with Age and Alzheimer's Disease Progression: Toward Mechanistic Insights and Accessible Lipid Biomarkers

Li-Huei Tsai, Ph.D.
Massachusetts Institute of Technology; Broad Institute

BRAIN AGING CONSORTIUM:

Choroid Plexus Aging and Alzheimer's Disease

Tony Wyss-Coray, Ph.D.
Stanford University

BRAIN AGING CONSORTIUM:

Establishing Isogenic Models of Human Neuron Aging and Pathways Relevant for Alzheimer's Disease

Andrew S. Yoo, Ph.D.
Washington University School of Medicine in St. Louis

Expanding Postmortem Brain Resources for Alzheimer's Disease in Diverse Populations

Sabina Berretta, M.D.
Harvard Medical School; McLean Hospital; Broad Institute

Thomas Blanchard, Ph.D., J.D.
University of Maryland School of Medicine

Identifying Sex-Aware X-Chromosome Targets for Preclinical Alzheimer's Disease

Rachel Buckley, Ph.D.
Massachusetts General Hospital; Brigham and Women's Hospital; Harvard Medical School

Menopause and Related Midlife Risk Factors and Their Impact on Pathology and Cognition: The WHIMSYAD Study

Rachel Buckley, Ph.D.
Massachusetts General Hospital; Brigham and Women's Hospital; Harvard Medical School

Midlife Autoantibody Profiles and the Risk of Late-Onset Alzheimer's Disease in Women

Yu Chen, Ph.D.
New York University

Viral Infections and Alzheimer's Disease Risk in Women

Yu Chen, Ph.D.
New York University

Stephen J. Elledge, Ph.D.
Brigham and Women's Hospital; Harvard Medical School

Restoring Brain Collection in New York City Through the Mount Sinai Neuropathology Brain Bank

John F. Crary, M.D., Ph.D.
Icahn School of Medicine at Mount Sinai

Plasma Proteins, Sex and Alzheimer's Disease: Proteome-Wide Analyses of the UK Biobank and Framingham Heart Study

P. Murali Doraiswamy, MBBS, FRCP
Duke University School of Medicine

Utility of Blood-Based Markers for Predicting Amyloid-Related Imaging Abnormalities and Their Course in Mild Cognitive Impairment and Alzheimer's Disease Subjects Undergoing Routine Clinical Treatment with Amyloid-Directed Antibodies

P. Murali Doraiswamy, MBBS, FRCP
Duke University School of Medicine

Bioinformatics Platform for Modeling Alzheimer's Progression (MAP-AD Platform)

Ali Ezzati, M.D.
University of California, Irvine

Alzheimer's Disease PET Imaging of Nonfibrillar Amyloid Beta Aggregates Using Azapeptide (AZP) Tracer

Samuel E. Gandy, M.D., Ph.D.
Icahn School of Medicine at Mount Sinai

Brigitte Guérin, Ph.D.
Université de Sherbrooke, Canada

William D. Lubell, Ph.D.
Université de Montréal, Canada

Shai Rahimipour, Ph.D.
Bar-Ilan University, Israel

Understanding Human Brain Resilience to Alzheimer's Pathology

Teresa Gomez-Isla, M.D.
Massachusetts General Hospital; Harvard Medical School

Identification and Validation of Plasma-Based Lipid Biomarkers for Early Alzheimer's Disease in the Unique, Primarily Hispanic, South Texas Population

Xianlin Han, Ph.D.

Tiffany F. Kautz, Ph.D.

Bernard Fongang, Ph.D.
The University of Texas Health Science Center at San Antonio

Utilizing Heterogeneous Mouse Models to Discover Mechanisms Underlying Caloric Restriction (CR) on Cognitive Outcomes

Catherine Kaczorowski, Ph.D.

Kelly Bakulski, Ph.D.
University of Michigan

FBAT-Equivalence Testing in the Presence of Model Uncertainty for the Cure Alzheimer's Fund Alzheimer's Genome Project™

Christoph Lange, Ph.D.

Niklas Hagemann, Ph.D.
Harvard T.H. Chan School of Public Health

Development of a FLAP-Targeted Neuroimmune PET Tracer for Imaging Microglia in Alzheimer's Disease

So Jeong Lee, Ph.D.
Massachusetts General Hospital; Harvard Medical School

Relationship Between Alzheimer's Disease Risk Score and Outcomes of Mild Repetitive Neurotrauma

Thomas W. McAllister, M.D.
Indiana University School of Medicine

Michael McCrea, Ph.D., ABPP
Medical College of Wisconsin; Wisconsin Institute of NeuroScience

Bridge Funding for a Younger Diverse Human Brain Bank to Investigate Early Alzheimer's Disease Pathology

Meaghan Morris, M.D., Ph.D.
Juan Troncoso, M.D.
Johns Hopkins University
Ling Li, M.D.
University of Maryland School of Medicine

Characterization of Alzheimer's Disease Molecular Biomarker Profiles Throughout the Pathobiological Continuum

Krista L. Moulder, Ph.D.
Washington University School of Medicine in St. Louis

Characterization of the Longitudinal Trajectories of the Synaptic Blood Marker Beta-Synuclein During Alzheimer's Disease Pathogenesis and Improvement of the Measurement Procedure

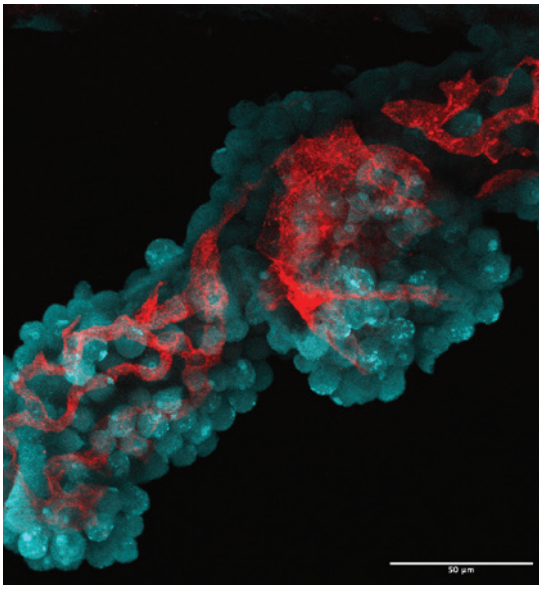
Patrick Oeckl, Ph.D.
German Center for Neurodegenerative Disease (DZNE), Germany
Markus Otto, M.D.
Martin-Luther-University Halle-Wittenberg, Germany

Cross-Species Analysis of Microglial States in Heterogeneous Alzheimer's Disease Progression

Towfique Raj, Ph.D.
Icahn School of Medicine at Mount Sinai
David Knowles, Ph.D.
Columbia University

Dynamic Investigation of Lymphatic Pathway Interconnections for Cerebrospinal Fluid Clearance of the Human Central Nervous System in Vivo Under Physiological Conditions of the Brain's Borders

Daniel S. Reich, M.D., Ph.D.
National Institute of Neurological Disorders and Stroke, National Institutes of Health
Lina C. Anderhalten, M.D., Ph.D.
National Institute of Neurological Disorders and Stroke, National Institutes of Health; Charité – Universitätsmedizin Berlin, Germany



The choroid plexus is a highly vascularized tissue that floats within the brain ventricles, produces cerebrospinal fluid (CSF), and forms the blood-CSF barrier. Epithelial cells (cyan) envelop a dense vasculature (red), forming a dynamic secretory interface that delivers a milieu of factors critical for brain homeostasis. Despite this central role, how the choroid plexus ages and how its dysfunction contributes to Alzheimer's disease remains poorly understood. Tracking newly synthesized choroid plexus proteins across brain, CSF, and blood opens new avenues to identify biomarkers, therapeutic targets, and molecular signatures of brain resilience.

Image attribution: Bhawika Sharma Lamichhane, Ph.D., from the lab of Tony Wyss-Coray, Ph.D., at Stanford University.

Expanding Deep South Inclusion in the UAB ADRC Neuropathology Core Brain Bank

Kirsten E. Schoonover, Ph.D.
Rati Chkheidze, M.D.
University of Alabama at Birmingham

Cognitively Healthy Nonagenarians in the Cross-Cohort Collaboration (CCC)

Sudha Seshadri, M.D.
The University of Texas Health Science Center at San Antonio

Digital Neuropathology Infrastructure for Precision Medicine in South Texas Hispanic Alzheimer's Disease and Alzheimer's Disease Related Dementias (AD/ADRD) Populations

Sudha Seshadri, M.D.
The University of Texas Health Science Center at San Antonio

A Three-Dimensional Tissue Model Linking Tau Tangles, Amyloid Beta and Microglial CD33-Isoform State to Gene- and Isoform-Expression Dysregulation

Hagen Tilgner, Ph.D.
Weill Cornell Medicine

A Temporally Aligned Multimodal Framework for Individualized Alzheimer's Disease Risk Assessment and Deep Learning-Driven Prognosis

Zheyu Wang, Ph.D.
Brian Caffo, Ph.D.
Johns Hopkins University

Reference Intervals for Plasma P-Tau217 in the Canadian Population

Cheryl Wellington, Ph.D.
Isabella DiBerardino, B.S.
University of British Columbia, Canada

▶ BIOLOGICAL RESEARCH MATERIALS: NEW ANIMAL AND CELLULAR MODELS, AND HUMAN SAMPLES

Investigating the Serial Pathologies Related to Plasma Biomarkers in NLFTaum/h Mice: A New Mouse Model Featuring Neurofibrillary Tangles as a Result of Rising Amyloid Beta without Microtubule-Associated Protein Tau (MAPT) Mutations

Frances Edwards, Ph.D.
John Hardy, Ph.D.
University College London, England

Engineering a Human Region-Specific System to Probe APOE-Linked Cellular Vulnerability and Resilience in Alzheimer's Disease

Mehdi Jorfi, Ph.D.
Massachusetts General Hospital; Harvard Medical School

Personalized Disease Prediction for Alzheimer's Disease Using Proteome Profiling: The EPIC4AD Study

Christina M. Lill, M.D., M.Sc.
University of Münster, Germany; Imperial College London, England
Lars Bertram, M.D.
University of Lübeck, Germany

Growth, Characterization and Distribution of a Neurodegenerative Disease-Focused Fibroblast/iPS Cell Bank to Support Molecular Models of Patient-Specific Variation with Validation in Matched Donated Brain Tissues

Derek H. Oakley, M.D., Ph.D.
Massachusetts General Hospital; Harvard Medical School

Characterization and Validation of Two Recently Created Sheep Models of Alzheimer's Disease in Preparation for Use as a Preclinical Pharmaceutical Testing Model

Russell G. Snell, Ph.D.
Natasha McKean, Ph.D.
University of Auckland, New Zealand

From Molecular Heterogeneity to Predictive Medicine: Transforming Neurodegenerative Disease Research Through Spatial Multi-Omics and Interpretable AI

Genevieve Stein-O'Brien, M.H.S., Ph.D.
Johns Hopkins University

Genes to Therapies™ (G2T) Research Models and Materials

Taconic Biosciences

Effect of APOE Genotype in a Novel Rat Model of Cerebral Amyloid Angiopathy

William Van Nostrand, Ph.D.
The University of Rhode Island

Decoding Protein Transport Across the Blood-Brain Barrier to Enable New Alzheimer's Therapies

Andrew Yang, Ph.D.
Gladstone Institutes; University of California, San Francisco

2025 Jeffrey L. Morby Prize for Exceptional Research

Andrew S. Yoo, Ph.D.
Washington University School of Medicine in St. Louis

Dissecting Alzheimer's Disease Phenotypes in Directly Reprogrammed Patient-Derived Neurons

Andrew S. Yoo, Ph.D.
Washington University School of Medicine in St. Louis

▶ EPIGENETIC FACTORS

CIRCUITS: Characterizing Epigenetic Biomarkers of Human Cognitive Aging

Lars Bertram, M.D.
University of Lübeck, Germany

Translational Research

▶ STUDIES OF NOVEL ALZHEIMER'S DISEASE GENES

Neural Stem Cell Rejuvenation for Symptomatic Alzheimer's Disease

Michael A. Bonaguidi, Ph.D.
Wynnie Nguyen, B.S.
University of Southern California

Multiomic and Functional Characterization of Soluble TREM2 Modifiers

Carlos Cruchaga, Ph.D.
Washington University School of Medicine in St. Louis

The Impact of Mutations in the Ligand-Binding Domain of CD33 on Alzheimer's Disease Pathogenesis

Ana Griciuc, Ph.D.
Massachusetts General Hospital; Harvard Medical School

Dissecting the Modulatory Roles of Interleukin-17 Receptor D in Alzheimer's Disease

Jun Huh, Ph.D.
Harvard Medical School

Elucidating the Therapeutic Potential of the Endo-Lysosome Pathway for Alzheimer's Disease

Jessica Young, Ph.D.
University of Washington

▶ STUDIES OF AMYLOID PRECURSOR PROTEIN AND AMYLOID BETA

RIN3 and BIN1 Interaction Connects Endosomal Pathology with Amyloid Pathology

Raja Bhattacharyya, Ph.D.
Massachusetts General Hospital; Harvard Medical School

Role of Stabilization of MAMs and MAM-associated Palmitoylated APP (MAM-palAPP) in Alzheimer's Disease

Raja Bhattacharyya, Ph.D.
Massachusetts General Hospital; Harvard Medical School

Understanding Amyloid Pathology—Multiomic Activity Imaging of Plaque Formation Dynamics (AmyMAP)

Frances Edwards, Ph.D.
University College London, England
Jörg Hanrieder, Ph.D.
University of Gothenburg, Sweden

Assessing the Role of APOE in Glial Lipid Droplet Metabolism and Function

Lance A. Johnson, Ph.D.
University of Kentucky

Structural Mimicry in Microbial and Antimicrobial Amyloids Connected to Neurodegenerative Diseases

Meytal Landau, B.Pharm, M.Sc., Ph.D.
Technion, Israel Institute of Technology, Israel; Deutsches Elektronen-Synchrotron (DESY), Germany

APP Gene Dose-Mediated Dysregulation of the Endolysosomal Network Acts to Compromise Synaptic Structure and Function Leading to Alzheimer's Disease in Down Syndrome

William C. Mobley, M.D., Ph.D.
University of California, San Diego

ADAM10 Cleavage of Amyloid Precursor Protein: Physiological Function in the Brain and Therapeutic Potential for Alzheimer's Disease

Jaehong Suh, Ph.D.
Massachusetts General Hospital; Harvard Medical School

BIN1 in Microglial Heterogeneity Along Alzheimer's Disease Progression and Microglial Function

Gopal Thinakaran, Ph.D.
University of South Florida

STUDIES OF TAU

ALZHEIMER'S DISEASE TAU CONSORTIUM:

Strain Replication in Mouse and Cell Models

Marc I. Diamond, M.D.
University of Texas Southwestern Medical Center

ALZHEIMER'S DISEASE TAU CONSORTIUM:

Post-Translational Modifications and Tau Ultrastructure; Impact of Amyloid Beta on Tau In Vivo

Karen E. Duff, Ph.D.
University College London, England

René Frank, Ph.D.
University of Leeds, England

ALZHEIMER'S DISEASE TAU CONSORTIUM:

Toxic Consequences of Early Tau Seeding

Bradley T. Hyman, M.D., Ph.D.

Rachel Bennett, Ph.D.
Massachusetts General Hospital; Harvard Medical School

ALZHEIMER'S DISEASE TAU CONSORTIUM:

The Role of Amyloid Beta-Induced Membrane Damage in Tau Pathology

Katherine Sadleir, Ph.D.

Robert Vassar, Ph.D.
Northwestern University Feinberg School of Medicine

ALZHEIMER'S DISEASE TAU CONSORTIUM:

Deep Mass Spectrometry Profiling of Tau Aggregates in Alzheimer's Disease and Other Tauopathies

Henrik Zetterberg, M.D., Ph.D.
University of Gothenburg, Sweden; University College London, England

Gunnar Brinkmalm, Ph.D.
University of Gothenburg, Sweden

Bridging the Gap: Sex-Specific Drivers of Senescence Underlying Neurodegeneration

Darren J. Baker, Ph.D.
Mayo Clinic, Rochester

Microglial Mitochondrial Dysfunction and Tau Toxicity in Alzheimer's Disease

George S. Bloom, Ph.D.

Andrés Norambuena, Ph.D.
University of Virginia

Cellular Vulnerability to Pathological Tau Protein Accumulation in Alzheimer's Disease

Mathieu Bourdenx, Ph.D.

Karen E. Duff, Ph.D.
University College London, England

Multiomics Characterization of Tau Pathology Onset and Its Relationship with Amyloid in the Human Hippocampus

Inma Cobos, M.D., Ph.D.
Stanford University

Identifying Mediators of Tau-Mediated Neuronal Necroptosis Using an Innovative In Vivo CRISPR Screen

Bart De Strooper, M.D., Ph.D.
VIB-KU Leuven Center for Neuroscience, Belgium; University College London, England

Engineered CAR-Macrophages to Clear Tau

Marc I. Diamond, M.D.
University of Texas Southwestern Medical Center

Mathew Blurton-Jones, Ph.D.
University of California, Irvine

Selective Vulnerability in Posterior Cortical Atrophy

John R. Dickson, M.D., Ph.D.

Bradley T. Hyman, M.D., Ph.D.
Massachusetts General Hospital; Harvard Medical School

Using Long-Read Sequencing to Investigate the MAPT Locus and Transcripts in Neurodegeneration

John Hardy, Ph.D.
University College London, England

Sleep Augmentation Reinforces Neural Computation, Enhances Cognition and Mitigates Neurodegenerative Disease Symptoms

Keith B. Hengen, Ph.D.

James McGregor, Ph.D.
Washington University in St. Louis

Tau-Induced Postsynaptic Dysfunction in Tauopathy Models

Karin Hochrainer, Ph.D.
Weill Cornell Medicine

Costantino Iadecola, M.D.
Weill Cornell Medical College

How Do Microglia Contribute to the Spread of Tau Pathology in Alzheimer's Disease?

Sarah C. Hopp, Ph.D.
University of Texas Health Science Center at San Antonio

Hypertension, Tau and Neurodegeneration

Costantino Iadecola, M.D.

Giuseppe Faraco, M.D., Ph.D.
Weill Cornell Medical College

Modulating the Levels of Tau-Seed Interactors to Treat Alzheimer's Disease

Cristian Lasagna-Reeves, Ph.D.
Indiana University School of Medicine

Characterization of Tau Pathology Heterogeneity Across the Alzheimer's Disease Spectrum

Niklas Mattsson-Carlgren, M.D., Ph.D.
Oskar Hansson, M.D., Ph.D.
Lund University, Sweden

Deciphering Phosphorylation-Dependent Tau Envelope Dysfunction to Prevent Alzheimer's Neurodegeneration

Richard J. McKenney, Ph.D.
University of California, Davis

Investigating the Role of Tau Protein in Neuronal Senescence

Miranda E. Orr, Ph.D.
Washington University School of Medicine in St. Louis

Define ADGRG1-Mediated Protective Microglial Response to Tau Tangles in Alzheimer's Disease

Xianhua Piao, M.D., Ph.D.
Jarin Tusnim, Ph.D.
University of California, San Francisco

Role of Tau in Presynaptic Dysfunction During the Cellular Phase of Alzheimer's Disease

Jeffrey Savas, Ph.D.
Northwestern University Feinberg School of Medicine

The Role of Planar Cell Polarity Proteins in Tau Oligomer-Induced Synapse Degeneration

Yimin Zou, Ph.D.
University of California, San Diego
Lulu Jiang, M.D., Ph.D.
University of Virginia

STUDIES OF APOLIPOPROTEIN E (APOE)

FLEMING APOE CONSORTIUM:

APOE Genotype-Specific Effects of Human Young Plasma on Cerebrovasculature and Alzheimer's Disease Pathology

Guojun Bu, Ph.D.
The Hong Kong University of Science and Technology

FLEMING APOE CONSORTIUM:

APOE4 Accelerates CD8 Exhaustion Via Glucocorticoid Signaling in Alzheimer's Female Carriers

Oleg Butovsky, Ph.D.
Vijay K. Kuchroo, D.V.M., Ph.D.
Brigham and Women's Hospital; Harvard Medical School

FLEMING APOE CONSORTIUM:

Cell Autonomous Roles of Protective APOE Variants in Microglia in Response to Amyloid Pathology

Michael Haney, Ph.D.
University of Pennsylvania

FLEMING APOE CONSORTIUM:

Investigating Potential Cell Autonomous Neuroprotection of APOE Protective Variants

David M. Holtzman, M.D.
Washington University School of Medicine in St. Louis

FLEMING APOE CONSORTIUM:

Modulation of Selective Neuronal Vulnerability in Alzheimer's Disease by Apolipoprotein E

Jean-Pierre Roussarie, Ph.D.
Boston University

FLEMING APOE CONSORTIUM:

APOE and Immune Checkpoint Regulation During Aging

Cheryl Wellington, Ph.D.
Phillip Domeier, Ph.D.
Marc Horwitz, Ph.D.
University of British Columbia, Canada

Investigating Lysosomal Mechanisms of Risk and Resilience in Alzheimer's Disease

Joel Blanchard, Ph.D.
Icahn School of Medicine at Mount Sinai

APOE in Choroid Plexus Function and Related Alzheimer's Disease Pathogenesis

Guojun Bu, Ph.D.
The Hong Kong University of Science and Technology

Mitochondrial Alzheimer's Risk Factors Control APOE Expression and Secretion

Victor Faundez, M.D., Ph.D.
Emory University

Unravelling Precision Hormone Therapy Using Menopause and Hormone Therapy Type Across APOE Genotype

Liisa Galea, Ph.D.
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Circuit Dynamics in APOE4 Mice

Ksenia Kastanenka, Ph.D.
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Neuroproteasomes Mechanistically Connect APOE Isoforms to Endogenous Tau Aggregation

Kapil V. Ramachandran, Ph.D.
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Elucidating the Interplay Between Alzheimer's Disease Genetic Variants Using Human iPSC-Derived Microglia-Like Cells

Giuseppina Tesco, M.D., Ph.D.
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Doo Yeon Kim, Ph.D.
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Elucidating the Protective Effects of APOE2 in the Presence of APOE4 Gene Allele in Animal Models

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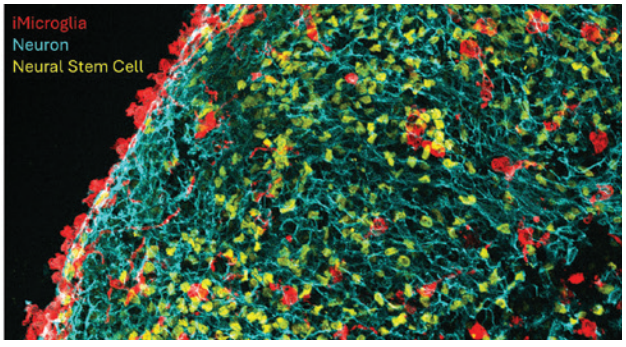

Model of human-induced pluripotent stem cell-derived brain organoids to investigate microglia-specific phenotypes in Alzheimer's disease.

Image attribution: Satish Tiwari from the lab of Florent Ginhoux, Ph.D., Singapore Immunology Network (SIgN), A*STAR.

STUDIES OF THE IMMUNE RESPONSE IN ALZHEIMER'S DISEASE

NEUROIMMUNE CONSORTIUM:

Examining the Impact of Peripherally Derived Human Macrophages in Alzheimer's Disease Pathogenesis

Mathew Blurton-Jones, Ph.D.
University of California, Irvine

NEUROIMMUNE CONSORTIUM:

Mechanisms Mediating Microglia Sensing of Peripheral Inflammation

Christopher K. Glass, M.D., Ph.D.
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NEUROIMMUNE CONSORTIUM:

Astrocyte Inflammatory Contributions to Alzheimer's Disease

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NEUROIMMUNE CONSORTIUM:

Effects of Peripheral Inflammation on Myeloid Cell Function in Alzheimer's Disease

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NEUROIMMUNE CONSORTIUM:

Impact of AD Polygenic Risk Score on Microglial Response to Peripheral Inflammation

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The Role of Astrocyte-Secreted Insulin-Like Growth Factor Binding Protein 2 (IGFBP2) in the Progression of Alzheimer's Disease

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Contributions of IL34 Signaling to Microglial Function and Alzheimer's Pathology in Mice

Staci Bilbo, Ph.D.
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Prenatal Inflammation Effects on Blood-Brain Barrier Function and Alzheimer's Disease-Related Pathologies Across the Lifespan

Alexandre Bonnin, Ph.D.
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A New Model of Microglia Genetic Perturbation in Vivo to Screen All Risk Factors Associated with Alzheimer's Disease

Oleg Butovsky, Ph.D.
Vijay K. Kuchroo, D.V.M., Ph.D.
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Mechanisms Regulating Neurovascular Function in Alzheimer's Disease

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Defining a Role for the MS4A Genes in Alzheimer's Disease

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VGF-Derived Peptide Therapy for Alzheimer's Disease: Studies of Mouse and Human TLQP-21 and its Receptor, C3aR1

Michelle E. Ehrlich, M.D.
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Tau and Amyloid Beta are Innate Immune Antimicrobial Peptides in the Brain

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Suppressing Neuroinflammation with a Targeted Protein Degradation Strategy in a Tauopathy Mouse Model

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Targeting Reactive Astrocytes AMPK Signaling to Suppress Inflammation in Alzheimer's Disease

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Microglial-Astrocytic Mechanisms of APOE3 Christchurch-Mediated Resilience to Tauopathy

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Sex-Specific Mechanisms of Neuroinflammation in the Alzheimer's Disease Hypothalamus

David M. Gate, Ph.D.
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Impact of DNA Damage-Mediated Stimulator of Interferon Genes (STING) Activation on Myelin Function in an Alzheimer's Disease Animal Model

Alban Gaultier, Ph.D.
University of Virginia

Multidimensional Profiling of TREM2-Mutated or APOE4-Mutated Microglia in Human Brain Organoids to Understand Dysregulated Microglia Neuronal Crosstalk in Alzheimer's Disease

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Endogenous Human Antibodies Associated with Alzheimer's Disease

Charles Glabe, Ph.D.
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Combinatorial Roles of EED and PICALM in Microglial Clearance Pathways in Alzheimer's Disease

Alison M. Goate, D.Phil.
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Investigating MEF2C Transcription Factor as a Therapeutic Target to Reprogram Pathological Microglial States in Alzheimer's Disease

Alison M. Goate, D.Phil.
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Investigating the Pathogenetic Role of Microglial MEF2C in Alzheimer's Disease Across the Lifespan: Developmental Priming vs. Adult Response to Injury

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Investigating Alzheimer's Disease-Associated Membrane Biology in Microglia and Neurons

Anna Greka, M.D., Ph.D.
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Beth Stevens, Ph.D.
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Precision Gene Therapy Targeting Microglia in Alzheimer's Disease

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Signaling Function of TREM2 Cleavage Products, Which Are Affected by Agonistic Antibodies to the Stalk Region

Christian Haass, Ph.D.

Kai Schlepckow, Ph.D.
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Antiviral Cytokine Signaling Promotes Neurodegeneration in Alzheimer's Disease

Jasmin Herz, Ph.D.
Washington University School of Medicine in St. Louis

Antiviral T Cell Infiltration to the Meninges and Brain Influences Neurodegeneration in Alzheimer's Disease

Jasmin Herz, Ph.D.
Washington University School of Medicine in St. Louis

2024 Jeffrey L. Morby Prize for Exceptional Research

David M. Holtzman, M.D.
Washington University School of Medicine in St. Louis

An Engineered Platform to Model the Yolk Sac-Brain Interactions in Alzheimer's Disease

Mehdi Jorfi, Ph.D.
Massachusetts General Hospital; Harvard Medical School

Mo Ebrahimkhani, M.D.
University of Pittsburgh

Role of CD8+ T Cell-Glial Interactions in Mediating Alzheimer's Disease Pathogenesis

Mehdi Jorfi, Ph.D.

Joseph Park, Ph.D.

Rudolph E. Tanzi, Ph.D.
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To Elucidate the Role of Memory T Cells as a Determinant of Age-Related Inflammation in Alzheimer's Disease

Susan Kaech, Ph.D.
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Semaphorin Regulation of Disease-Associated Microglia in Alzheimer's Disease

Tae-Wan Kim, Ph.D.
Columbia University

Role of Checkpoint Molecules TIM-3 and LAG-3 in Microglial Function in Alzheimer's Disease

Vijay K. Kuchroo, D.V.M., Ph.D.
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Tracking Inflammation and Repair at the Choroid Plexus in Alzheimer's Disease

Maria K. Lehtinen, Ph.D.

Christian Lagares Linares
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Do Classical Complement Activation and the Route of Administration of Anti-Amyloid Antibodies Contribute to Vascular Side Effects Known as Amyloid-Related Imaging Abnormalities?

Cynthia A. Lemere, Ph.D.
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The Role of Interferon-Induced Transmembrane Protein 3 (IFITM3) and Gamma-Secretase in Microglia

Yueming Li, Ph.D.
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Mechanisms of Astrocyte-Derived Lipid Toxicity in Alzheimer's Disease

Shane A. Liddelow, Ph.D.
New York University

Elucidating the Role of CLEC7A in Tau-Mediated Neurodegenerative Disease

John R. Lukens, Ph.D.
University of Virginia

Meningeal Regulatory T Cells (Tregs) in Individuals with Versus without Alzheimer's Disease

Diane Mathis, Ph.D.
Harvard Medical School

Investigating the Association Between Clonal Hematopoiesis and Alzheimer's Disease

Cameron McAlpine, Ph.D.
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Elucidating Mechanisms Driving the Compromised Balance Between Mitophagy and cGAS-STING-Initiated Inflammation Toward a Treatment for Alzheimer's Disease

Per Nilsson, Ph.D.
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Evandro F. Fang, Ph.D.
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Centenarian Microglia Reveal a Neprilysin-Dependent Mechanism of Resilience in Alzheimer's Disease

Joseph Park, Ph.D.

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Probing the Molecular Underpinnings of G Protein-Coupled Receptor ADGRG1 Mediated Protective Microglial Responses to Alzheimer's Disease

Xianhua Piao, M.D., Ph.D.
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Extracellular ATP is a Key Factor in Promoting Alzheimer's Disease Neuroinflammation

Paola Pizzo, Ph.D.
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Anna Lisa Giuliani, Ph.D.
University of Ferrara, Italy

Specificity of T-Cell Responses in Autosomal Dominant Alzheimer's Disease (ADAD)

Naresha Saligrama, Ph.D.
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Ventricular Ependymal Cells: A Novel Neural-Immune Signaling Axis During Aging and Neurodegeneration

Dorothy P. Schafer, Ph.D.
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How Body-Brain Inflammatory Signals Via Border Macrophages Alter the Neuroimmune Landscape and Drive Alzheimer's Pathology

Beth Stevens, Ph.D.
Boston Children's Hospital; Harvard Medical School; Broad Institute

Helena Barr, Ph.D.
Broad Institute; Boston Children's Hospital

Non-Invasive Modulation of Microglia Gene Expression Using Peripherally Administered Antibody Conjugates

Peter M. Tessier, Ph.D.
Colin F. Greineder, M.D., Ph.D.
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Dissecting Microglial State Dynamics in Alzheimer's Disease

Li-Huei Tsai, Ph.D.
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Understanding the Dynamic Lipid-Immunometabolome of Protective and Risk Alzheimer's Microglia

Rik van der Kant, Ph.D.
Amsterdam University Medical Center, The Netherlands

T-Cell Modulation of Microglia to Treat Alzheimer's Disease

Howard L. Weiner, M.D.
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Cancer-Derived Extracellular Vesicle microRNAs as Systemic Modulators of Alzheimer's Disease

Stephen T.C. Wong, Ph.D.
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Decipher the Astrocyte Cell-Surface Proteome in Alzheimer's Disease

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Junmin Peng, Ph.D.
St. Jude Children's Research Hospital

STUDIES OF ALTERNATIVE NEURODEGENERATIVE PATHWAYS

BRAIN ENTRY & EXIT CONSORTIUM:

Central Nervous System Fluid Homeostasis and Waste Clearance in Alzheimer's Disease Characterized by MRI

Helene Benveniste, M.D., Ph.D.
Yale School of Medicine
David Xianfeng Gu, Ph.D.
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BRAIN ENTRY & EXIT CONSORTIUM:

Human Three-Dimensional Neurovascular Interaction and Meningeal Lymphatics Models with Application to Alzheimer's Disease

Se Hoon Choi, Ph.D.
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Roger D. Kamm, Ph.D.
Massachusetts Institute of Technology

BRAIN ENTRY & EXIT CONSORTIUM:

How Does Vascular Fatty Acid Metabolism Regulate the Pathophysiology of Alzheimer's Disease?

Richard Daneman, Ph.D.
University of California, San Diego

BRAIN ENTRY & EXIT CONSORTIUM:

Meningeal Mast Cell Control of Cerebrospinal Fluid Dynamics in Homeostasis and Alzheimer's Disease

Jonathan Kipnis, Ph.D.
Washington University School of Medicine in St. Louis

BRAIN ENTRY & EXIT CONSORTIUM:

Neuroinflammation at the Choroid Plexus in Alzheimer's Disease

Maria K. Lehtinen, Ph.D.
Harvard Medical School; Boston Children's Hospital
Liisa Myllykangas, M.D., Ph.D.
University of Helsinki, Finland

BRAIN ENTRY & EXIT CONSORTIUM:

Does Subarachnoid Lymphatic-like Membrane (SLYM) Failure Compromise Glymphatic Clearance in Alzheimer's Disease?

Maiken Nedergaard, M.D., D.M.Sc.
University of Rochester; University of Copenhagen, Denmark

BRAIN ENTRY & EXIT CONSORTIUM:

High-Resolution Magnetic Resonance Imaging of the Brain Borders

Daniel S. Reich, M.D., Ph.D.
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BRAIN ENTRY & EXIT CONSORTIUM:

Biochemical and Functional Analysis of Cerebrospinal Fluid and Lymph Following Changes in Brain Fluid Dynamics

Laura Santambrogio, M.D., Ph.D.
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MICROBIOME CONSORTIUM:

Harnessing Diet-Microbe Interactions to Prevent Alzheimer's Disease Pathogenesis

Laura M. Cox, Ph.D.
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MICROBIOME CONSORTIUM:

The Role of Gut Microbial Metabolism in Tau-Mediated Neurodegeneration

David M. Holtzman, M.D.
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MICROBIOME CONSORTIUM:

Temporal Relationships Between Gut Dysbiosis, Brain Amyloid Beta Metabolism and Microglia Cell Activation Following Antibiotic Treatment

Sangram S. Sisodia, Ph.D.
The University of Chicago

MICROBIOME CONSORTIUM:

Microbial Profiling of Human Brain and Gut Microbiomes in Alzheimer's Disease

Rudolph E. Tanzi, Ph.D.
Nanda Kumar Navalpur Shanmugam, Ph.D.
Massachusetts General Hospital; Harvard Medical School

MICROBIOME CONSORTIUM:

Interaction of the Microbiome with Astrocytes and Amyloid Pathology

Robert Vassar, Ph.D.
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BMP Lipid Metabolism as a Therapeutic Target for Lysosomal Function and Alzheimer's Disease Pathogenesis

Monther Abu-Remaileh, Ph.D.
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Alzheimer's Disease Pathophysiology Alters the Level of Electrical and Chemical Synapse Coupling in the Network of GABAergic PV+ Interneurons Early in Disease Course

Srdjan D. Antic, M.D.
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Disentangling the Role of Intracranial Arteriosclerosis in Alzheimer's Disease

Daniel Bos, M.D., Ph.D.
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Geert Jan Biessels, M.D., Ph.D.
University Medical Centre Utrecht, The Netherlands
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Scaling the Divide in Alzheimer's Disease: An Integrated Molecular, Cellular and Network-Level Study

Marc Aurel Busche, M.D., Ph.D.
Samuel Harris, Ph.D.
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Neuroprotective Effects of the Exercise Hormone Irisin in Alzheimer's Disease

Se Hoon Choi, Ph.D.
Christiane Wrann, D.V.M., Ph.D.
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Restore Meningeal Lymphatic Drainage to Alleviate White Matter Damage and Cerebral Amyloid Angiopathy in a Model of Alzheimer's Disease

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Mayo Clinic, Jacksonville

Decoding Microbial Products Modulating Alzheimer's Disease—Toward Precision Postbiotics Treatment

Eran Elinav, M.D., Ph.D.
Weizmann Institute of Science, Israel; DKFZ, Germany

Turning Up Mitophagy to Blunt Alzheimer's Tau Pathologies

Evandro F. Fang, Ph.D.
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Air Pollution and Alzheimer's Disease Risk Interact with Premature Aging of Neural Stem Cells and Apolipoprotein E Alleles

Caleb E. Finch, Ph.D.
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Bridging Myelin and Vascular Dementia: Oligodendrocyte Stress Responses in Aging and Cerebrovascular Disease

Margaret E. Flanagan, M.D.
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In Vivo Models for Golgi Fragmentation and the Molecular Pathogenesis of Alzheimer's Disease

Samuel E. Gandy, M.D., Ph.D.
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Oligodendroglial Dynamics and Myelination in Alzheimer's Disease

Erin M. Gibson, Ph.D.
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Understanding the Mechanism Underlying Vaccination for Alzheimer's Disease

Charles L. Greenblatt, M.D.
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Linking Sleep Dysfunction to Tau-Related Degeneration Across Alzheimer's Disease Variants

Lea Tenenholz Grinberg, M.D., Ph.D.
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The Role of Calcium Homeostasis in Axonal Spheroid Formation in Alzheimer's Disease

Jaime Grutzendler, M.D.
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Proteomic Signatures of Cerebrovascular Neuropathology in Down Syndrome with Alzheimer's Disease

Elizabeth Head, Ph.D.
Vivek Swarup, Ph.D.
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Deciphering and Restoring Computational Setpoints in Alzheimer's Disease Through Sleep-Enhanced Network Homeostasis

Keith B. Hengen, Ph.D.
Washington University in St. Louis

Sleep Reinforces Homeostatic Setpoints in Neural Activity and Mitigates Neurodegenerative Disease

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Novel Artificial Intelligence (AI) Decodes Aging Neurons

Andrew J. Holbrook, Ph.D.
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Theodore Zwang, Ph.D.
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Lowering Microglial Lipid Burden Via Perilipin-2 (PLIN2) Modulation in Alzheimer's Disease

Lance A. Johnson, Ph.D.
University of Kentucky

Morphological, Electrophysiological and Transcriptional Characterization of Single Neurons from Resilient and Susceptible Models of Human Alzheimer's Disease

Catherine Kaczorowski, Ph.D.
Shannon Moore, Ph.D.
University of Michigan

Neuronal Mechanisms Driving Synapse Loss in Alzheimer's Disease

Martin Kampmann, Ph.D.
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The Multiomic Roadmap to Neuronal Senescence in Alzheimer's Disease

Takahisa Kanekiyo, M.D., Ph.D.
Yingxue Ren, Ph.D.
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Interneuron Therapy for Alzheimer's Disease

Ksenia Kastanenka, Ph.D.
Massachusetts General Hospital; Harvard Medical School

Neuroprotective T Cell-Based Therapy for Alzheimer's Disease

Jonathan Kipnis, Ph.D.
Washington University School of Medicine in St. Louis

Characterizing Gut Bacteriome-Mycobiome Synergy in Correlation to Amylin-Amyloid Beta Antimicrobial Synergy in Alzheimer's Disease (AD) in AD Mouse Models

Deepak Kumar Vijaya Kumar, Ph.D.
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Astrocyte Network Structure and Function in Alzheimer's Disease

Shane A. Liddelow, Ph.D.
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Circadian Perturbations of the Vasculome and Microgliome in Alzheimer's Disease

Eng H. Lo, Ph.D.
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Elucidating the Role of Emerging Genomic Stress Sensors in Alzheimer's Disease

John R. Lukens, Ph.D.
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Circadian Desynchrony, Glial Dysfunction and Alzheimer's Disease Pathogenesis

Erik S. Musiek, M.D., Ph.D.
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Dysregulation of Signaling on Post Synaptic Density Scaffolds in Alzheimer's Disease

Alexandra C. Newton, Ph.D.
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Developing Cell-Type-Specific Enhancer-AAV Vectors to Characterize and Restore Amyloid Beta- and Tau-Dependent Circuit and Cognitive Deficits in Humanized Alzheimer's Mouse Models

Jorge Palop, Ph.D.
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Evaluating the Contribution of TDP-43 Dysfunction and Cryptic Mis-Splicing to Alzheimer's Disease Pathogenesis

Leonard Petrucelli, Ph.D.
Mayo Clinic, Jacksonville

Unraveling Mixed Pathology and Its Impact on the Proteome in Alzheimer's Disease

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Protection Against Alzheimer's Disease with Longevity-Promoting Intervention 17a-Estradiol

Christian Pike, Ph.D.
Bérénice A. Benayoun, Ph.D.
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2-Deoxyglucose and Its Analogs as Novel Therapeutics to Build Resilience to Alzheimer's Disease

Rajiv R. Ratan, M.D., Ph.D.
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Theodore J. Lampidis, Ph.D.
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The Impact of Soluble Dietary Fiber on Tau-Mediated Neurodegeneration in a Mouse Model of Tauopathy

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Pre-Clinical Testing of CDK4/6 Inhibitors as a Therapeutic Strategy in Alzheimer's Disease Using Alzheimer's Disease Tauopathy Mouse Model

Peter Sicinski, M.D., Ph.D.
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Identifying the Sex-Specific Roles of the Gut Microbiome-Brain Axis in a Mouse Model of Amyloid Beta Amyloidosis

Sangram S. Sisodia, Ph.D.
The University of Chicago

Role of Psychosocial Stress in Alzheimer's Disease

Filip Swirski, Ph.D.
Icahn School of Medicine at Mount Sinai

Role of Annexin A6-Mediated Membrane Repair in Alzheimer's Pathology

Robert Vassar, Ph.D.
Achint Kaur, B.S.
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Role of Platelet-Derived Factors in Ameliorating Alzheimer's Disease Pathology

Saul Villeda, Ph.D.
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A Multimodality Study on the Lipid Molecular Basis of Obesity and Its Roles in Regulating Alzheimer's Pathogenesis for Developing Potential Targeted Interventions

Stephen T.C. Wong, Ph.D.
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Noncoding Translation Feedback Loop in Alzheimer's Disease

Xuebing Wu, Ph.D.
Columbia University

Drug Discovery and Enabling Technologies

▶ DRUG SCREENING AND LEAD DRUG EVALUATION PROJECTS

Development of Small Molecule Inhibitors of Cholesterol 25-hydroxylase

Anil Cashikar, Ph.D.
Bahaa Elgendy, Ph.D.
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Validation and Characterization of Compounds Modulating Neuroinflammation and Amyloid Beta Uptake in Microglial Cells

Ana Griciuc, Ph.D.
Luisa Quinti, Ph.D.
Massachusetts General Hospital; Harvard Medical School

Exploring Novel Drug Candidates for Alzheimer's Disease Through Integrative Pathway Analysis and Validation in 3D Cellular Models

Doo Yeon Kim, Ph.D.
Luisa Quinti, Ph.D.
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Development and Mechanism of Autophagy Activators for Treatment of Alzheimer's Disease

Yueming Li, Ph.D.
Memorial Sloan Kettering Cancer Center

Identification and Development of CD33 Inhibitors and Pre-RNA Splicing Modulators

Subhash Sinha, Ph.D.
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▶ DRUG DELIVERY AND ENABLING TECHNOLOGIES

Developing Brain-Targeted AAVs Using Human Blood-Brain Barrier Receptors

Leszek Lisowski, Ph.D., MBA
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Matthieu Drouyer, Ph.D.
Children's Medical Research Institute, Australia

Gloria González-Aseguinolaza, Ph.D.
University of Navarra, Spain

Preclinical and Clinical Drug Development and Trials

▶ PRECLINICAL DRUG DEVELOPMENT

SON-Dependent Nuclear Speckle Rejuvenation: A Novel Proteostasis-Based Therapeutic Strategy for Alzheimer's Disease

Xu Chen, Ph.D.
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Brain-Penetrant Complement Blocking VHHs to Reduce Neuroinflammation in Alzheimer's Disease

Maarten Dewilde, Ph.D.
KU Leuven, Belgium

Wioleta Zelek, Ph.D.
Cardiff University, Wales, United Kingdom

Sex-Biased Toll-Like Receptor 7 (TLR7) Signaling in Demyelination and Its Inhibition by Small Molecules

Li Gan, Ph.D.

Subhash Sinha, Ph.D.
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Targeting Neuroinflammation with Nasal Administration of Anti-CD3 Monoclonal Antibody to Treat Alzheimer's Disease

Rafael M. Rezende, Ph.D.
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Non-Invasive Delivery of IL-2 to the CNS for Local Expansion of Regulatory T Cells and Prevention of Neurodegeneration in Tauopathy

Peter M. Tessier, Ph.D.
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David M. Holtzman, M.D.
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Preclinical Analysis of Synaptogyrin-3 Oligonucleotides to Target Tauopathy

Patrik Verstreken, Ph.D.
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Characterization and Optimization of CNS-Penetrant HDAC11-Selective Inhibitors in Alzheimer's Disease Models

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▶ CLINICAL TRIALS

A Proposal to Evaluate the Effect of Bacillus Calmette-Guérin Vaccination on Alzheimer's Disease Development

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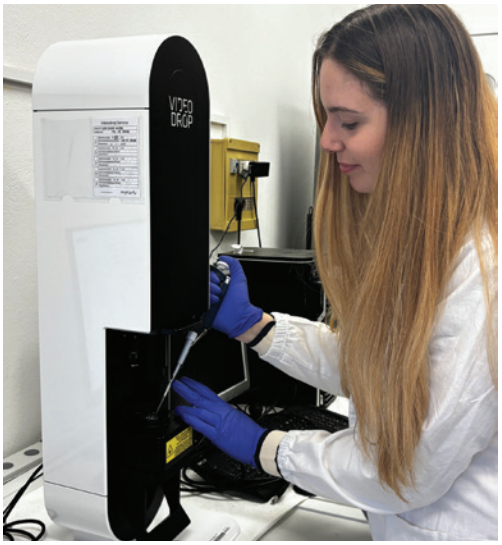
Continuous Monitoring and Enhancement of Glymphatic Flow in Human Sleep

Seattle Neurosound Solutions



A researcher analyzes extracellular vesicles using a VideoDrop system, capturing a precise moment in Alzheimer's research. This work reflects her focus on understanding how cellular communication contributes to the disease. It also embodies resilience: the persistence to explore complex, often invisible processes, driven by the shared hope of improving the lives of patients and their families.

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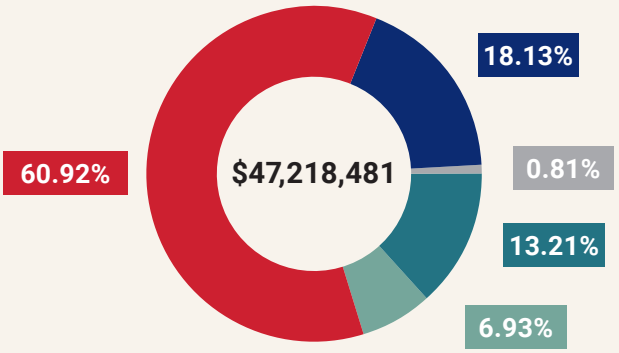
2025 Source and Use of Funds

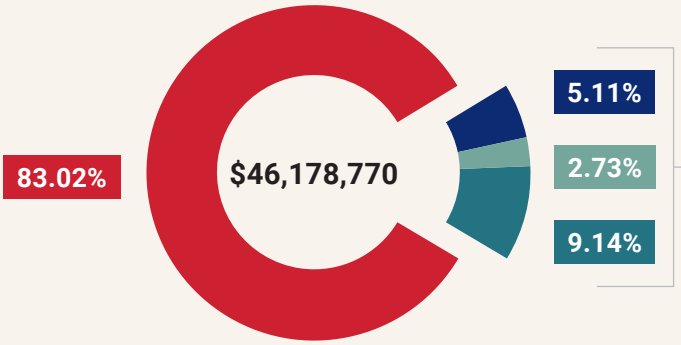
In 2025, our Total Fundraising (TFR) was $47,218,481. Cure Alzheimer's Fund received $8,558,855 in Foundation Fundraising (FFR) from our Founders, Board of Directors, Trustees, and a core group of other donors to support our operations. Our donors contributed $38,659,626 in General Fundraising (GFR) to support our research programs.

SOURCE OF FUNDS



- Individuals and Families **$28,768,959**
- Founders/Board of Directors/Trustees/ Core Group of Other Donors for FFR **$8,558,855**
- Corporations **$380,643**
- Foundations **$6,238,308**
- Trusts/Estates **$3,271,716**

USE OF FUNDS



- Research Distributions and Support **$38,338,893**

Operating Expenses Paid by Founders/Board of Directors/Trustees/ Core Group of other Donors for FFR:

- Fundraising **$2,357,449**
- Management and General **$1,262,143**
- Programs **$4,220,285**

Source: Internal records

2025 Financials

(Year ended December 31, 2025)

Statement of Financial Position

Assets	
Current Assets:	
Cash and cash equivalents	$5,367,416
Pledges receivable, current portion	4,000,000
Investments	25,565,254
Prepaid expenses and other current assets	321,471
Total current assets	35,254,141
Pledges receivable, less current portion, net	5,622,053
Investments	2,138,842
Leasehold improvements, net	5,068
Right-of-Use Asset, net	149,480
Total Assets	$43,169,584
Liabilities and Net Assets	
Current Liabilities:	
Current portion of operating lease payable	$76,940
Accounts payable	363,629
Research grants payable	4,812,010
Accrued payroll and related	739,590
Total current liabilities	5,992,169
Long-term Liabilities:	
Operating lease payable, less current portion	65,190
Total long-term liabilities	65,190
Total liablilities	6,057,359
Net Assets:	
Without donor restrictions	26,004,933
With donor restrictions	11,107,292
Total net assets	37,112,225
Total Liabilities and Net Assets	$43,169,584

Statement of Activities

Revenue and Support:	
Contributions	$49,364,961
Donated stock	5,646,939
Special events, net of direct expenses	335,288
Investment income, net	1,079,619
Total revenue and support	56,426,807
Expenses:	
Program:	
Research distributions and support	38,338,893
Other program expenses	4,220,285
Total program expenses	42,559,178
Management and general	1,262,143
Fundraising	2,357,449
Total expenses	46,178,770
Change in net assets	10,248,037
Net Assets, beginning of year	26,864,188
Net Assets, end of year	$37,112,225

Source: Audited Financial Statements available at **curealz.org/2025financials**

Our People

BOARD OF DIRECTORS

HENRY F. McCANCE

Chair, Board of Directors, and Founding Board Member

Chairman Emeritus, Greylock Partners

Partner, Fenway Sports Group

Trustee, McCance Family Foundation

JACQUELINE C. MORBY

Founding Board Member

Senior Advisor, TA Associates

Chair, Morby Family Charitable Foundation

(Deceased May 2026)

PHYLLIS RAPPAPORT

Treasurer

Founding Board Member

Chair, Phyllis and Jerome Lyle Rappaport Charitable Foundation

MEG SMITH

Chief Executive Officer, Cure Alzheimer's Fund

TIM ARMOUR

Former President and Chief Executive Officer, Cure Alzheimer's Fund

RICHARD BIRNBAUM

Chairman and Director, Rick Sharp Alzheimer's Foundation

BRITTANY BOWLEN

Board of Directors, Boys & Girls Clubs of Metro Denver

Board of Visitors, Duke University Fuqua School of Business

MARK FAGGIANO

Founder and Former CEO, TaxJar, acquired by Stripe

MARY GREENHILL CAGLIERO

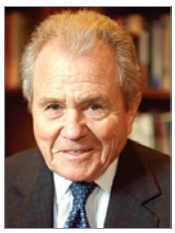
Member, Board of Directors

KUMAR MAHADEVA

Founder and Former CEO, Cognizant Technology Solutions

CHRISTINE VILLAS-BOAS

President, Michel & Claire Gudefin Family Foundation



ROBERT F. GREENHILL*

Founder, Greenhill & Company

**Emeritus*

TRUSTEES

KATHLEEN ARNOLD
Trustee, Fleming Foundation

JONO BACON
Founder, Community Leadership Core

CAROL BAXTER
Baxter Family Foundation

JAMES BEERS

ELLEN BERK

MICHAEL BERK
Managing Director, TA Associates

ANOOSHEH BOSTANI
Director, Alfred E. Mann Charities

JEFF AND BLAKELEY BURATTO

THOMAS CLARKE
CEO, The Clarke Companies

SHARI CROTTY
Trustee, The Crotty Family Foundation

KAREN FRIEND, PH.D., ACPS
Senior Researcher and Evaluator, Pacific Institute for Research and Evaluation; Adjunct Professor, Brown University Department of Public Health

CHRISTINA KOHNEN
Trustee, Kohnen Family Foundation

JEANNE LESZCZYNSKI
Director of Public Health, Associate Professor of Pathology, UMass Medical School, retired

EDIE AND JOHN RANDALL

NANCY AND BRYAN RUEZ

LINDSEY SIMON
CEO and Founder, Simon Compliance

ADMINISTRATION

JO ANTONELLIS
Chief Financial Officer

TAMMY AWTRY, PH.D.
Science Communicator

KYRSTEN CONOVER
Development Coordinator, Operations

ALLISON DeCRUZ
Director of Planned Giving

DANNY HARPER
Vice President, Development

HEIDI HENDERSON
Controller

MORGAN HERMAN
Executive Vice President, Development

LAURA HILL
Development Operations Assistant

LAINIE HOLCOMB
Manager, Community Fundraising

NATASHA HUSSAIN, PH.D.
Executive Vice President, Research Management

ANDREW KOEMETER-COX, PH.D.
Vice President, Research Management

KYLE LIOLIOS
Donor Relations Associate

LAUREL LYLE
Vice President, Board Relations and Development Operations

JEVON MANSFIELD
Director, Donor Relations and Events

LORI MARCHETTI
Accounting Supervisor

KAREN MURRAY
Human Resources Manager

CHRISTINA NOVAK
Senior Philanthropic Advisor, Institutional Relations

JORDANA PEREIRA
Executive Coordinator

LORI PRESCOTT
Accounting Specialist II

LISA RAND
Vice President, Marketing and Communications

ERIN REID
Philanthropic Advisor, Leadership Gifts

CAITLIN SAIA
Senior Director, Grant Administration

CHARLES SEAKS, PH.D.
Director, Grant Management

NIKKI SENGSAVANH
Senior Director, Development Operations

SHARON SEVRANSKY
Development Coordinator

KAELA S. SINGLETON, PH.D.
Director, Grant Management

JOHN SLATTERY
Senior Vice President, Philanthropic Partnerships

MEG SMITH
Chief Executive Officer

HAILEY STOLTENBERG
Administrative Assistant, Research Management

DOROTHY VACARO
Gift Processing Coordinator

ELIZA WHYTE
Office Operations Manager

EMANUELA ZAHARIEVA RAPPOPORT, PH.D.
Science Communicator

Working Together for a Cure

The Power of Community

Alzheimer's disease inflicts a terrible toll, but it also reveals the strength and resilience of a community determined to find a cure. Each person's experience is unique, yet together our resilience fuels progress—raising funds, awareness, and hope. By supporting research in ways that reflect their passions and abilities, our donors move us closer to a future without Alzheimer's.

The Ralston Family

JESSE RALSTON

Jesse Ralston's support of Cure Alzheimer's Fund is rooted in personal experience and a belief in the power of research to change the future. An entrepreneur himself, he was drawn to CureAlz for its research-driven, entrepreneurial approach and its focus on advancing meaningful progress.

Jesse's father, David, lived with Alzheimer's before passing in 2024. Jesse remembers him as "a loving, funny, and giving person," someone who would share anything he had and who found joy in life's simplest moments, from music to family meals around a table he built himself. His generosity of spirit has helped to shape the way Jesse moves through the world.

Today, Jesse carries that spirit forward. Through a monthly contribution from his donor-advised fund, he is making a steady and meaningful investment in research, honoring his father's memory while helping to build a better future for his own daughters and for families everywhere.

Jesse and his family are guided by a sense of hope. "The large number of projects focusing on solving the Alzheimer's problem," he shares, "make me believe that a solution will be possible in the foreseeable future."

Driving Progress Through Corporate and Foundation Support

Corporations and foundations play a vital role in the fight against Alzheimer's disease. Their commitment to giving goes beyond writing a check. Through fundraising in their community, matching employee gifts, payroll giving programs, and more, their leadership and generosity help turn determination into progress and hope into action.

RICK SHARP ALZHEIMER'S INITIATIVE AT CURE ALZHEIMER'S FUND

Richard L. Sharp—known to all as Rick—was a visionary leader devoted to his family and friends whose impact extended far beyond the boardroom. He believed that success came from surrounding himself with exceptional people and a willingness to transform industries.

As CEO of Circuit City, Rick led the nation's first big-box electronics retailer, reshaping the consumer shopping experience. He later brought that same entrepreneurial spirit to new frontiers—as a founding investor and CEO of Crocs, and as the founder of CarMax, the innovative used car superstore that revolutionized an industry.

Sherry and Rick Sharp

Beyond his professional accomplishments, he shared 46 years of love and partnership with his high school sweetheart, Sherry. They raised two wonderful daughters and delighted in their four grandchildren. When diagnosed with Alzheimer's disease in 2010, Rick faced it with courage, optimism, and determination to advance research for a cure.

Following his passing, Sherry, along with family and friends, carried forward his legacy by establishing the Rick Sharp Alzheimer's Foundation (RSAF). Donating millions of dollars to CureAlz, the RSAF has funded research projects that bring us closer every day to understanding—and ultimately curing—this devastating disease. In 2025, the creation of the Rick Sharp Alzheimer's Initiative at Cure Alzheimer's Fund deepened this shared mission, ensuring that Rick and Sherry's spirit of vision, determination, and hope continue to inspire progress for years to come.

THE BERGER FAMILY

Steve Berger lived a life defined by generosity, humor, and deep devotion to the people he loved. To his family, he was the center of countless memories filled with laughter, adventure, and unwavering encouragement. A devoted husband, father, and grandfather, Steve shared 55 wonderful years of marriage with his beloved wife, Dee, and took great pride in raising his sons and cheering on his grandchildren. Whether coaching sports, planning family trips, or simply telling one of his many jokes, he had a way of bringing people together and making every moment brighter.

Steve approached life with energy and curiosity. He loved skiing, biking, and especially time spent with family and friends at Lake Tahoe, where so many of their favorite memories were made. His charisma, kindness, and larger-than-life personality left a lasting impression on everyone fortunate enough to know him.

His family remembers Steve as a man who gave freely of his time, his encouragement, and his heart. In his memory and honor, they support Cure Alzheimer's Fund in the hope that continued research will help others live fuller, healthier lives.

Steve Berger

From left: Carole, Eileen (Mom), Sherie, and Robert "Bob" (Dad)

BERKOWITZ FAMILY FOUNDATION

"Family was the most important thing to our dad, Robert," said Sherie Wolpert. "He was kind, generous, curious, energetic, and loving. Although he had a very successful career, he also ran the New York Marathon in his 50s, learned to bake breads and cakes after he retired, took guitar lessons, and helped everyone who was in need.

"Bit by bit, he lost the ability to have meaningful conversations that we all cherished. Among all the things Alzheimer's took from him, this is what we miss most.

"We were eager to support an organization that is prudent with donations and were impressed that 100% of general gifts directly support CureAlz's research program.

"Our dad believed so much in helping others that we are certain he would be proud to know that our family foundation was supporting Alzheimer's research so that future generations will not need to suffer as he did."

Turning Passions into Fundraising

As our fundraising partners, CureAlz Heroes not only champion research through their support, but also inspire hope and progress. From bake sales to road races, livestreams and everything in between, any passion can become a fundraising activity. Visit **give.curealz.org/hero** to get started.

BALD MOVE 24-HOUR GROUNDHOG DAY MOVIE MARATHON

"What if we watched the movie 'Groundhog Day' on repeat for 24 hours on Groundhog Day?"

In 2016, A. Ron Hubbard and Jim Jones, co-founders and co-hosts of the Bald Move podcast, turned a ridiculous idea into reality by livestreaming themselves watching the Bill Murray classic for 24 hours straight. Bald Move produces podcasts about film and television, so the event was perfectly suited for their movie-loving community.

What began as a fun challenge soon became something more meaningful. A. Ron suggested turning the movie marathon into a fundraiser, and a few years later they began supporting Cure Alzheimer's Fund to honor A. Ron's grandfather and aunt, who both passed away from the disease.

The Groundhog Day Movie Marathon is now an annual tradition. In 2025, Bald Move watched "The Lord of the Rings" and "The Hobbit" movies, raising a record $50,000 for Alzheimer's research.

ALEXA BURTON – EAST EGG COMMUNITY FUN RUN

Each summer since 2022, sisters Alexa (20) and Neve (18) Burton have transformed their neighborhood into a center of hope with the East Egg Community Fun Run, a fundraiser benefiting Cure Alzheimer's Fund. The annual race honors their grandmother's memory in the place she once called home.

"The event has become a staple in our community. People are really excited to come together every year," said Alexa. "I've made so many connections with neighbors who've lost loved ones to Alzheimer's. The race has truly allowed us to find community in the face of a disease that can be so isolating for families and caregivers."

In 2025, the East Egg Community Fun Run raised a record-breaking $5,650. That summer Alexa interned with the Cure Alzheimer's Fund Heroes Program, which supports community fundraisers like hers across the country. In her role, she helped strengthen fundraising efforts that directly advance the CureAlz mission.

Alexa, now a rising junior at Brown University, has raised money for Cure Alzheimer's Fund since middle school. She ran the Sri Chinmoy Marathon (Congers, New York) in 2022 as well as participated in multiple half-marathons, and has launched countless other initiatives to raise awareness for Alzheimer's research.

Alexa Burton

COURTNEY IVERSON – MORELS & MEMORIES

Courtney launched the Morels & Memories Mushroom Hunt in 2016 to honor her mother, Heidi Vanderlinde, who was diagnosed with early-onset Alzheimer's at age 49. Inspired by Heidi's love of the outdoors, plants, and sunshine, Courtney turned her passion for foraging into a fundraiser. The event began with 25 friends—but now more than 250 participants gather at her family farm in Watertown, Minnesota, each year.

Foragers compete for prizes like the first, largest, and most morels found, guided by local experts and celebrity foragers. The day concludes with a morel tasting, comfort food, live music, and a silent auction.

"It's amazing how my world has expanded just because I tried to do something when you're told there's nothing you can do," Courtney shared.

Six years after her mother's passing, Courtney continues to honor her legacy through this beloved annual event, which marked its 10th year in May 2025. Since its founding, Morels & Memories has raised more than $127,000 for Alzheimer's research.

Where Thoughtful Planning Creates Meaningful Change

A lasting legacy is built on hope, compassion, and impact. By partnering with Cure Alzheimer's Fund through a planned gift, donors help empower visionary scientists to pursue the bold research breakthroughs needed to transform the future of Alzheimer's.

DIANE DAUTOFF

Diane Altman Dautoff's commitment to advancing Alzheimer's research is rooted in love, experience, and a deep sense of responsibility for future generations. Her mother, Marion, lived with Alzheimer's for nearly two decades, yet remained, as her family remembers, "a happy woman" who greeted others with warmth, humor, and hugs almost to the end of her life. Witnessing her mother's long journey shaped Diane's determination to be part of the solution.

Together with her husband, Stuart, Diane has chosen to turn that experience into meaningful action. In addition to her generous philanthropic support, Diane participates in research studies herself, recognizing that progress depends on individuals willing to contribute their time for the benefit of others. Most recently, Diane discovered that she has amyloid in her brain and is in the process of learning what that might mean for her own future. This quiet, heroic act of generosity reflects Diane's hope for a better future for her daughter, grandchildren, and families everywhere.

From left: Marion Altman and Diane Altman Dautoff

Through her thoughtful decision to include Cure Alzheimer's Fund in her estate plans, Diane ensures that her impact will extend far beyond her lifetime. Her legacy is one of compassion, foresight, and an enduring commitment to helping others.

You can create a lasting legacy...

by including Cure Alzheimer's Fund in your will or trust, through a charitable gift annuity, or by naming CureAlz as a beneficiary of a retirement or financial account, insurance policy, or donor-advised fund.

To explore planned giving options that best meet your needs, contact us at **legacy@curealz.org**.

With Your Partnership, We Will Find a Cure

There are many ways to be part of the solution to find a cure. With your support, we can truly make a difference in our understanding of Alzheimer's and bring hope to the millions of individuals and their families who are affected by this disease.

Our Board of Directors, Trustees, and a core group of other donors direct their donations to our overhead expenses so that **100% of general donations support our research program.**

MAKE AN IMPACT

- Make a one-time or recurring gift online by credit card, PayPal, or Venmo. Visit **give.curealz.org/annualreport**.
- Mail a check payable to Cure Alzheimer's Fund.
- Recommend a gift to Cure Alzheimer's Fund through your donor advised fund (DAF).
- Donate by telephone. Our business hours are 9 a.m.–5 p.m. ET.
- Support CureAlz with stocks, mutual fund shares, or other appreciated assets while potentially reducing your tax burden. Gifts may be sent electronically via wire transfer. Please contact Laurel Lyle at **LLyle@curealz.org**.
- Make a tax-free gift to CureAlz through a qualified charitable distribution (QCD): if you are age 70½ or older, you can donate up to $111,000 directly from your individual retirement account (IRA) in 2026.
- Become a community fundraiser. Visit **give.curealz.org/hero** or email **hero@curealz.org** to connect with a member of our team.

Cure Alzheimer's FUND

34 Washington St.
Suite 230
Wellesley Hills, MA 02481
Info@CureAlz.org
781-237-3800

DONATE TODAY

Scan the QR code, visit **give.curealz.org/annualreport**, email us at **info@curealz.org**, or call **781-237-3800**.

Cure Alzheimer's Fund is a "doing business as" name for the Alzheimer's Disease Research Foundation, a 501(c)(3) public charity with federal tax ID #52-2396428.

Cure Alzheimer's FUND
34 Washington St., Suite 230
Wellesley Hills, MA 02481

CureAlz.org

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