

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.

Annual Report 2024

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Dear Friends,

When we founded Cure Alzheimer's Fund in 2004, I could not have imagined everything we would accomplish.

Beginning with three founding families and the work of a Dream Team of researchers led by Dr. Rudy Tanzi, our funding has led to remarkable discoveries changing the world's understanding of Alzheimer's disease.

Thanks to all of you, 2024 was a wonderful milestone year for Cure Alzheimer's Fund as we marked our 20th year funding research to accelerate the end of Alzheimer's disease. We distributed a record \$30 million toward our research program in 2024 and, as of the writing of this report, reached a total of \$229 million in research funding since our inception. What a tremendous achievement, all made possible by your unwavering support and commitment to our mission.

Our team comes together around our shared mission and the principles with which we pursue it—principles that are recognized by the broader philanthropic world and that will hold us in good stead as we face new challenges in our fight against Alzheimer's disease. In 2024, we received a 100% Perfect Score from Charity Navigator, an accomplishment earned by less than 1% of all rated nonprofits, on top of our 13th consecutive 4-star rating. This extraordinary accomplishment recognizes our commitment to transparency, financial responsibility and good governance-ensuring that 100% of general donations continue to support our research program.



Henry F. McCance

We share this annual report at a time of much uncertainty surrounding funding by the National Institutes of Health (NIH), the largest funder of Alzheimer's research in the world. The NIH has slowed or halted funding authorized and, in some cases, already awarded by Congress. It also announced a policy change capping reimbursement for indirect costs at 15%, a significant reduction of \$4 billion from prior levels, with huge potential to impact the research and operations of labs and their institutions. As of the writing of this letter, the policy change sits in the courts, and the impact of this NIH upheaval on our scientists, their labs and their research is not yet known.

The well of innovation from which biotech and pharma draw upon relies on a steady flow of great new knowledge and ideas. Future drugs and treatments can exist only if precompetitive research happens first, investigating key biological questions and providing proof of concept for new targets, delivery mechanisms, therapeutic approaches and more. For-profit entities cannot and will not support it. Any one drug that reaches the market is the result of decades of building block ideas and projects, each contributing necessary components to a successful treatment. CureAlz supports, identifies and tests the most promising new ideas, and NIH support then provides further demonstration of their value and feasibility.

"CureAlz supports, identifies and tests the most promising new ideas, and NIH support then provides further demonstration of their value and feasibility."

As we look back on the research funded since our inception, all that we have accomplished together is truly remarkable. Yet we are acutely aware of how much work remains. On behalf of 50 million worldwide who have been diagnosed with Alzheimer's diseaseand an estimated 150 million who have cognitive decline not yet diagnosed-CureAlz remains steadfast in our commitment to understand the causes and pathology of Alzheimer's disease so that researchers throughout the world can use the discoveries in their efforts to find therapies leading to a cure.

IN GRATITUDE

I am personally grateful to my fellow members of our Board of Directors and am pleased to welcome five new directors to the Cure Alzheimer's Fund Board. Brittany Bowlen, Mark Faggiano, Mary Greenhill Cagliero, Kumar Mahadeva and Christine Villas-Boas bring proven leadership expertise and success in business, and share a personal desire to eliminate Alzheimer's disease. We will all benefit from their insights and passion for our mission. I want to thank our Trustees and other core donors who join our Board in supporting all of the overhead costs of Cure Alzheimer's Fund. Together, with our dedicated staff, we recognize the thousands of researchers in labs around the world who are working tirelessly to understand this very complex disease.

And, of course, my deepest gratitude goes to each of you—our generous donors who make all of our research possible. Your support has fueled two decades of scientific progress, and your continued commitment gives us great optimism and confidence that, in the near future, the research we fund will lead to prevention and to effective therapies for each of the stages of Alzheimer's disease.

Together we will continue this crucial work until Alzheimer's disease is nothing but a memory.

Sincerely,

Henry F. McCance Chair, Board of Directors

A MESSAGE FROM OUR CHIEF EXECUTIVE OFFICER

In 2004, the founders of Cure Alzheimer's Fund came together because they were frustrated with the slow pace of progress. They were determined to accelerate research, make bold bets and eradicate the disease. Twenty years later we celebrate the wins, mourn those we have lost, and remain inspired and determined by their vision.



Meg Smith

I have spent much of 2024 traveling the country talking to families affected by Alzheimer's disease, many of whom are desperately seeking therapies and all hopeful for a cure. Like me, they are glad for our 20 years of progress but impatient that progress does not yet mean solutions for their loved ones. I came to share new discoveries and knowledge with them, but I was humbled by what I learned from them: the places a newly diagnosed patient is determined to see, the daughter holding on to her memories of her father's wisdom, the husband celebrating his wife of 40 years speaking his name, the wife trying to keep her husband at home. I am honored that these families shared their stories with me, and by their trust in Cure Alzheimer's Fund and the research we support to accelerate an end to this disease.

So much has been accomplished over the last 20 years. We've identified gene variants that can increase or decrease your risk of Alzheimer's disease. We are beginning to understand the brain's immune system and to discover the impact of the composition of the gut microbiome on brain pathology. Biomarkers for diagnosis and staging now allow us to identify people with the disease, while new therapies can affect disease progression. Scientists funded by CureAlz have been at the forefront of groundbreaking findings. Our 325 amazing, top-tier scientists from 18 countries

collaborate, challenge and encourage one another for the benefit of science. Many of these scientists bring expertise in different specialties and come together through our consortia model to focus on a specific emerging and promising area of science to have the greatest impact.

In 2024, the CureAlz research program hit a recordbreaking \$30 million in research distributions and support of an extraordinary range of promising science to benefit the Alzheimer's community. Since our inception in 2004, we have deployed more than \$229 million to further research and support our mission.

At this moment, poised for breakthroughs thanks to new technologies and the great discoveries we have enabled, our field and all of biomedical science face enormous challenges. Trust in science has been undermined, and as a society we have lost sight of all the lives saved by careful use of the scientific method and resulting validated discoveries. Research in the United States is funded through private funders like CureAlz as well as public funders like the National Institutes of Health, the world's largest funder of biomedical research. The uncertainty around public funding in 2025, as of the writing of this annual report, threatens to slow the trajectory of progress we have worked so hard to achieve

The scientists we support, along with their institutions and colleagues, are under immense pressure. We are facing the potential loss of an entire generation of future discovery—young scientists in academia have always been underpaid relative to their peers in forprofit industry, and many will lose their jobs altogether under new policy mandates. Ongoing and new federally funded research has slowed and in some cases ceased due to funding uncertainty. This situation demonstrates the fragility of the system and the important role that private funders like CureAlz and supporters like you play in continuing this important and necessary work.

So, we roll up our sleeves and push forward-more determined and committed to fueling the momentum we have gained over the last 20 years. We are significantly closer to effective therapies and a cure than we were 20 years ago, but closer is not close enough. I remain inspired by the vision set by our founders in 2004. Their commitment and passion still guide us today. Most important are the more than 78,000 supporters who have joined CureAlz to support research since 2004. These significant accomplishments are your accomplishments. I hope you are as proud of the progress we have made together as I am. I am truly grateful for your continued support and the confidence you have in the CureAlz team to get us even closer to ending this disease. Thank you for joining CureAlz in the fight against Alzheimer's disease.

"In 2024, the CureAlz research program hit a record-breaking \$30 million in research distributions and support of an extraordinary range of promising science to benefit the Alzheimer's community."

Warmly,



Alzheimer's disease has no cure. Yet.

7.2
MILLION

Americans living with Alzheimer's disease

\$384

Projected cost of Alzheimer's and other dementias to the United States in 2025

12

MILLION

Americans providing unpaid care for a family member/ friend with dementia

Source: Alzheimer's Association, 2025 Alzheimer's Disease Facts and Figures

Research is the only path to a cure.

Since 2004, Cure Alzheimer's Fund has taken a unique approach to funding research with the highest probability of preventing, slowing or reversing Alzheimer's disease.



Fund high-risk, high-reward research with the potential to accelerate a cure or diseasemodifying treatment.



Require funded researchers to collaborate and share data.



Identify and support investigators who will contribute to the field's knowledge base with bold hypotheses and well-crafted experimental plans.



Fund scientists
quickly throughout
the year by
eliminating red tape
and streamlining
application
requirements.



Utilize teams of leading scientists to guide scientific direction, review grant proposals and assess the research portfolio.

Enabling the world's leading scientists to explore bold ideas and make game-changing discoveries.

325 **RESEARCHERS CONSORTIA COUNTRIES FUNDED FUNDED INSTITUTIONS**

We can do this because of you.

78,000+

\$219,000,000+

DONORS

GENERAL DONATIONS TO SUPPORT OUR RESEARCH PROGRAM

Together we are making an impact.

927

RESEARCH GRANTS

RESEARCH FUNDS DISTRIBUTED

1,434

RESEARCH PAPERS PUBLISHED

120,133 RESEARCH PAPER CITATIONS

Total grants provided to researchers from Cure Alzheimer's Fund 2018-2022

(Latest data available)

RESULTED

Extraordinary follow-on funding from NIH/NIA

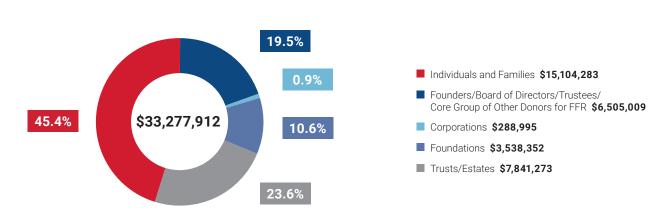
7.5 The return of follow-on funding from NIH/NIA 2018–2024

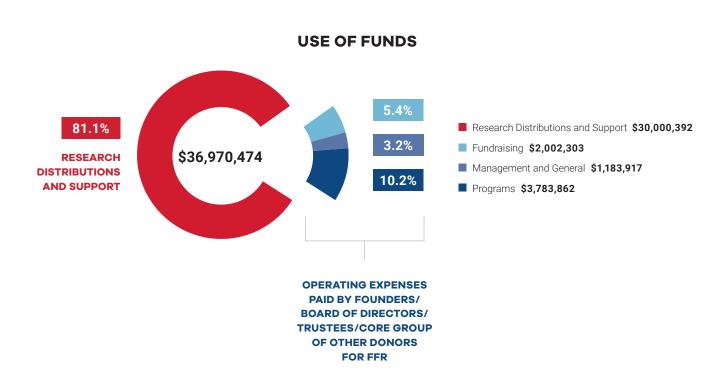
All CureAlz figures above reflect inception in 2004 through Dec. 31, 2024.

2024 Source and Use of Funds

In 2024, our Total Fundraising (TFR) was \$33,277,912. Cure Alzheimer's Fund received \$6,505,009 in Foundation Fundraising (FFR) from our Founders, Board of Directors, Trustees and a core group of other donors to support our operations. \$26,772,903 in General Fundraising (GFR) was received to support our research programs.







Statement of Financial Position

Assets	
Current Assets:	
Cash and cash equivalents	\$3,138,491
Contributions receivable	309,739
Pledges receivable, current portion	1,350,000
Investments	23,255,145
Prepaid expenses and other current assets	246,040
Total current assets	28,299,415
Pledges receivable, less current portion, net	47,994
Equipment and leasehold improvements, net	
	7,964
Right-of-Use Assets, net Total Assets	230,858 \$28,586,231
Total Assets	\$20,300,231
Liabilities and Net Assets	
Current Liabilities:	
Current portion of operating leases payable	\$74,028
Accounts payable	167,083
Research grants payable	600,901
Accrued payroll and related	730,551
Total current liabilities	1,572,563
Total current liabilities	1,372,303
Long-term Liabilities:	
Operating leases payable, less current portion	149,480
Total long-term liabilities	149,480
Total liablilities	1,722,043
Net Assets:	
Without donor restrictions	25,165,313
With donor restrictions	1,698,875
Total net assets	26,864,188
Total Liabilities and Net Assets	\$28,586,231
Statement of Activities	
Revenue and Support:	
Contributions	\$24,922,509
Donated stock	5,629,724
Special events, net of direct expenses	238,791
Investment income, net	
	1,282,683 32,073,707
Total revenue and support	32,073,707
Expenses:	
Program:	
Research distributions and support	30,000,392
Other program expenses	3,783,862
Total program expenses	33,784,254
rotai program expenses	33,764,234
Management and general	1,183,917
Fundraising	2,002,303
Total expenses	36,970,474
Change in net assets	(4,896,767)
Net Assets, beginning of year	31,760,955
Net Assets, end of year	\$26,864,188

Source: Audited Financial Statements

Statement of Cash Flows

Cash Flows from Operating Activities:	
Cash received from:	
Contributions	\$32,968,174
Investment income	969,120
Total receipts	33,937,294
Cash paid for:	
Research distributions and support	(30,117,918)
Salaries and related expenses	(4,578,920)
Professional fees	(632,046)
Gift processing fees	(177,074)
Occupancy expenses	(196,398)
Other expenses	(1,471,727)
Total expenditures	(37,174,083)
Net cash used by operating activities	(3,236,789)
Cash Flows from Investing Activities:	
Purchase of leasehold improvements	(8,688)
Proceeds from sale of investments	5,934,905
Purchase of investments	(4,763,747)
Net cash provided by investing activities	1,162,470
Net Decrease in Cash and Cash Equivalents	(2,074,319)
Cash and Cash Equivalents, beginning of year	5,212,810
Cash and Cash Equivalents, end of year	\$3,138,491

Alzheimer's Genome Project™

In 2005, Cure Alzheimer's Fund launched the first largescale, family-based study of the human genome specific to Alzheimer's, the Alzheimer's Genome Project™ (AGP), under Dr. Rudolph E. Tanzi's expert leadership.

AGP is a cornerstone of CureAlz research, advancing our understanding of the causes of Alzheimer's disease and bringing us closer to a cure. Constantly incorporating new technologies and approaches, AGP continues to uncover and illuminate the genetic drivers of the disease, pointing toward new lines of biological investigation and therapeutic targets.

The Origin of the Alzheimer's Genome Project

During the 1980s and 1990s, Dr. Tanzi and others identified the first three early-onset familial Alzheimer's genes, all implicating amyloid beta as a root cause of the disease. Given the technology at the time, the groundbreaking endeavor spanned many challenging years—and other biological evidence suggested that additional, as-yet unidentified factors had important roles in disease development and progression. "We only had the three early-onset genes that I had found with others and the APOE late-onset gene, found at Duke," remarked Dr. Tanzi.

Across biomedical research, drugs are most likely to succeed in clinical trials if they target biological pathways identified through human genetic evidence of disease association. But with such limited evidence, the development of novel Alzheimer's disease therapies stalled.

In the early 2000s, however, a synergy of impactful scientific advances presented a path forward to discovering new disease-associated genes. New technology enabled entire genomes to be scanned, the Human Genome Project mapped out the 20,000-

plus protein-coding human genes, and computing power made the wealth of information accessible. Recognizing



Rudolph E. Tanzi, Ph.D. Chair, Cure Alzheimer's Fund Research Leadership Group

the opportunity, Dr. Tanzi spearheaded AGP with an ambitious goal: to identify all genes affecting Alzheimer's risk to point the field at the proteins and pathways that matter in Alzheimer's. "[Before AGP,] somebody could just pick a protein and say, yeah, I think this protein is involved in Alzheimer's, and then study it their entire life and get nowhere because that protein didn't matter. If you want to know how to do something, first you have to know what matters, and genetics tells you what matters," explained Dr. Tanzi. AGP's wealth of genetic data would thus point drug developers toward the most promising targets for eventual clinical testing.

Key Discoveries and Advances

Since its inception, AGP has identified dozens of new gene variants associated with increased risk or resilience to Alzheimer's across sex, APOE status and in diverse populations. The discoveries made by AGP have contributed to more than 160 scientific publications, advancing the entire field's knowledge of the disease. AGP's groundbreaking work was recognized in 2008 when it was listed among Time magazine's Top 10 Medical Breakthroughs, solidifying its approach and ongoing discoveries.

AGP pioneered the use of family-based genome-wide association studies (GWAS) to scan the genomes of Alzheimer's patients in search of new Alzheimer's genes—and in 2008 found the first one involved with

neuroinflammation in the brain, known as CD33. "That's important because CD33 was the first gene that implicated innate immunity in the brain and microglia, which are now probably the most studied brain cell in the entire field," shared Dr. Tanzi.

We now know that as the sticky bundles of amyloid beta build up in the brain, it is the function of the innate immune cells—the microglia—to surround the bundles and clear them away. In Alzheimer's disease, the microglia do not adequately perform their clearance function, allowing amyloid beta to continue to accumulate and trigger the tau tangles inside nerve cells. Dysfunctional microglia also release inflammatory factors and interplay with other cell types to promote inflammation in the brain. This leads to 10 times more nerve cell death

Before the discovery of CD33 as the first innate immune Alzheimer's risk gene, the field primarily viewed Alzheimer's as a disease of neuron dysfunction alone; with this finding, microglia and the other cell types in the brain became part of the Alzheimer's story. Over time, additional GWAS studies identified many more genes involved with the brain's immune response. "Now, of the nearly 100 Alzheimer's genes known, two-thirds of them are microglial genes," Dr. Tanzi pointed out. These genetic studies have inspired new drug development and clinical research targeting microglia and neuroinflammation.

Further advances in DNA sequencing technology allowed AGP to expand its scope beyond the original GWAS approach that relied on preset chips that could enable scanning only of a defined set of genes. These chips yielded important findings about the genes the chipmaker selected for inclusion, but left much genetic information unread. As AGP has matured, it has taken an unbiased approach, scanning the entire somatic genome and discovering risk variants in coding and regulatory DNA that had previously gone unexamined. AGP achieved this by carrying out the first whole genome sequencing of Alzheimer's families in 2012. In addition,

"Our mantra from day one was early prediction, early detection and early intervention. We know Alzheimer's pathology happens years in advance of clinical symptoms. We can't wait for the brain to be failing before we do something about it."

-RUDOLPH E. TANZI, PH.D.

AGP continuously integrates additional, larger datasets of entire whole-genome sequences from biobanks in the United States and abroad, broadening the applicability of its findings by strengthening the diversity of its study population and the power of its statistical analytics.

Powered by the wealth of genetic information from the whole-genome sequencing datasets, AGP grew its analysis to hundreds of times more genetic variants for their association with Alzheimer's disease. To make sense of large-scale genetic data of this magnitude, AGP developed novel statistical tools to pinpoint those variants that robustly associated with the disease. In 2020, employing these new statistical approaches, AGP produced the first large, family-based whole-genome sequencing analysis for sex-specific associations with Alzheimer's disease. This study showed major differences in risk for men and women. The ZBTB7C gene was found to have the strongest association with Alzheimer's. Its variants conferred a high risk in women of developing the disease—while those same variants conferred disease protection in men. Given that twothirds of Alzheimer's patients are women, and given the disease's disproportionate impact on women that traditionally has been attributed to their longer lifespan, AGP's research indicates that genetic factors play a significant role beyond age alone.

Continuing its in-depth, whole-genome sequencing approach in 2021, AGP discovered rare variants associated with Alzheimer's found in less than 1% of the population. While the previously found common variants

were immune-related and altered risk weakly, the newly uncovered 13 rare genetic variants affected pathways related to synaptic function and neuroplasticity and had a strong impact on disease risk, generating new questions about Alzheimer's etiology that still are under investigation. The loss of synapses closely correlates with the severity of Alzheimer's cognitive symptoms, but a definitive genetic connection to synapse health had previously remained elusive. With its discovery, AGP established the first genetic link between Alzheimer's disease and synapse loss, identifying key targets in synapse function for biological investigation and therapeutic targeting.

By adding additional patient samples from more ethnicities along with those of European descent, AGP identified 16 new cohort-specific Alzheimer's disease-associated genes in 2025. This highlights the importance of studying diverse populations and further expanding the understanding of genetic risk factors to benefit all Alzheimer's patients.

Collaborative Studies

The successes of AGP transcend its genetic association studies for Alzheimer's disease. The discoveries are a foundation for collaborative efforts with other investigators to further functionally characterize the Alzheimer's genes identified by AGP, and to deepen understanding of how the consequences of our genes change during our lifetime. Using the revolutionary Alzheimer's in a Dish cell culture model, AGP investigates the genes' functional consequences. Partnering with other CureAlz investigators, Dr. Tanzi's AGP team has in recent years supported investigation of disease-associated changes in gene expression, interrogated the potential role of repetitive DNA sequences within Alzheimer's risk genes and developed new algorithms to deepen GWAS analytics from

association toward causality. Dr. Tanzi and AGP also share the most promising variants with Cure Alzheimer's Fund investigators and the greater Alzheimer's research community. These collaborations between genetic discovery and biological validation contribute to better understanding of how these genes affect amyloid accumulation, tau pathology, synapse function, inflammation and neurodegeneration.

Predictive Models

A major advancement in AGP's efforts is the application of machine learning models to enhance early diagnosis and prediction of Alzheimer's disease. By combining genetic, proteomic and metabolomic data with electronic health records, AGP is developing polygenic risk models capable of predicting an individual's likelihood of developing Alzheimer's and the likely age of onset. These predictive models are critical for early intervention strategies, potentially allowing at-risk individuals to take preventive measures.

"Our mantra from day one was early prediction, early detection and early intervention. We know Alzheimer's pathology happens years in advance of clinical symptoms. We can't wait for the brain to be failing before we do something about it," concluded Dr. Tanzi.

Looking Ahead

As AGP celebrates its 20th anniversary, its commitment to uncovering the genetic makeup of Alzheimer's disease and to translating those findings into therapeutic targets and polygenic risk scores remains steadfast. At the forefront of Alzheimer's research, AGP unites its wealth of genetics data with a collaborative ethos that knits together investigators across Cure Alzheimer's Fund, and together brings us closer to a future in which Alzheimer's has a cure.

Research Areas of Focus

Cure Alzheimer's Fund set a record for scientific funding in 2024. We deployed \$30 million for research purposes, an 8% increase from the previous record set in 2023 (\$27.8 million). This total represents funds in support of 120 grants and 137 named investigators.

A fundamental principle of CureAlz's approach to accelerating science to benefit the Alzheimer's disease (AD) community is to provide funding where other entities will not, and prioritize returns measured in scientific progress, not potential profits. Thus, Cure Alzheimer's Fund continues to prioritize target identification for Alzheimer's science research over proprietary drug development, with most distributions in 2024–92% of awards—directed to early-stage research.

Most funding went to translational research projects and a large fraction—approximately 24%—of all funded studies are on the neurodegenerative pathways in Alzheimer's disease. Under the right circumstances, CureAlz will continue to fund a great idea as it moves downstream closer to the clinic and patients; drug development efforts in this vein represent approximately 6% of funding in 2024. We funded a healthy balance of renewing projects and entirely new or follow-on projects as part of our portfolio.

Basic Research: Early Stage

Drug Development Process

Foundational (22%)

\$6,340,778

Research focused on problems or topics that haven't yet been defined with clarity. Topics include genetic risk factors, epigenetic factors, biomarkers, diagnostics, studies of risk and resilience, and new animal and cellular models of Alzheimer's disease.

Translational (70%)

\$20,361,238

Research aimed at translating results from foundational research into approaches that directly benefit humans. Topics include studies of novel Alzheimer's disease genes, amyloid precursor protein (APP), amyloid beta, tau, Apolipoprotein E (APOE), immune response, microbiome and alternative neurodegenerative pathways.

Drug Discovery and Enabling Technologies (3%)

\$805,000

Research aimed at developing potential treatments and approaches that could alleviate or prevent Alzheimer's disease.

Preclinical and Clinical Drug Development and Trials (5%)

\$1,524,640

Drugs and other therapies are further validated and optimized to maximize their chance of success in human clinical trials.

NOTE: Excludes research materials and scientific meeting support.

The evident progress and promising discoveries enabled by this funding are more important than these numbers. The incredible successes our researchers have had in publishing their impactful work is a sign that we are making the right funding decisions to help patients and their families who desperately need solutions.

Science Collaboration

Collaboration and information sharing are core principles of Cure Alzheimer's Fund, fostering a cooperative environment among the researchers we support. By working together across subdisciplines and institutions, our funded scientists leverage each other's discoveries and insights, accelerating progress in Alzheimer's research. These research partnerships play a crucial role in deepening our understanding of the disease and leading to potential solutions.

Consortia Model

CureAlz supports new high-risk, high-potential standalone grants while also supporting larger-scale investigations within specific areas of the science. The consortia model empowers these initiatives with an expanded level of collaboration. To read additional detail about each consortium, please visit **CureAlz.org/the-research/consortia/**.

ALZHEIMER'S DISEASE TAU CONSORTIUM

Neurofibrillary tau tangles emerge and propagate in the Alzheimer's brain at the same time that cognitive symptoms emerge. Tau's role in Alzheimer's disease (AD) and how it is induced by amyloid beta pathology are essential to understanding this disease. This consortium is investigating the initiation of tau pathology, neuron vulnerability and many other areas of AD tau biology.

■ BRAIN ENTRY AND EXIT CONSORTIUM

The brain is a remarkable but fragile organ that requires constant flows of nourishment in and debris out, but it has limited ability to renew itself if damaged by infection. A complex system of checkpoints controls the passage of materials between the brain and the rest of the body; the Brain Entry and Exit Consortium is investigating how these structures and the fluids that circulate among them function together to maintain health.

■ FLEMING APOE CONSORTIUM

APOE4 is the strongest negative genetic risk factor for late-onset sporadic Alzheimer's disease identified to date, whereas APOE2 is protective. Founded in 2017, this consortium is examining the many pathways in which the APOE protein plays a role to determine the mechanisms of its large impact on disease risk and progression.

■ MICROBIOME CONSORTIUM

The gut microbiome includes trillions of microorganisms, including bacteria, fungi and viruses, that live in the digestive tract and play an essential role in maintaining our health. Changes to composition of the gut microbiome can alter the state of our brains. This consortium is interrogating links between the gut microbiome and Alzheimer's disease.

■ NEUROIMMUNE CONSORTIUM

Our immune system, including the neuroimmune system of the brain, responds to both pathogens and everyday biological debris. The response must be proportionate and appropriate to the trigger, or else beneficial inflammation can turn damaging. The Neuroimmune Consortium is studying how the brain optimally recognizes and responds to AD pathology.

Advisory Groups

The collaborative efforts at Cure Alzheimer's Fund include groups of researchers working together to guide research distributions. 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 the field of Alzheimer's disease. 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

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VIB-KU Leuven, Belgium; University College London, England

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Nancy Ip, Ph.D.

The Hong Kong University of Science and Technology

Jonathan Kipnis, Ph.D.

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Christoph Lange, Ph.D.

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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

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

Rigiang 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

Rudolph E. Tanzi, Ph.D. Chair

Massachusetts General Hospital; Harvard Medical School

*New member

■ SCIENTIFIC ADVISORY BOARD (SAB)

The role of the SAB is to provide 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

Karen Reeves, M.D.

Ligand Pharmaceuticals

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

Looking Forward

Cure Alzheimer's Fund entered 2025 with a strong portfolio of compelling scientific ideas and, as the year has progressed, that portfolio has only grown. Our Research Leadership Group—made up of leading scientists in the field—continues to guide us in making strategic funding decisions and identifying high-priority areas for investigation. The study of aging across its many aspects is a priority for 2025.

Alzheimer's disease does not happen without aging. While the connection between aging and Alzheimer's has long been clear, aging itself is poorly understood and not well-defined. Not everyone ages the same way, at the same rate, or experiences the same kind of cognitive decline. Each of our cells, organs and systems ages individually as well as together; new discoveries and technologies allow us to investigate the changes that come with age in astonishing new ways. Understanding what drives healthy aging is critical to identifying what is normal and what may be a function of disease.

Many biological changes linked to aging influence brain health. For instance, chronic, low-grade inflammation in the body is a common feature of aging. Scientists now know that the body's immune system interacts with the brain, and chronic inflammation increases disease risk. Exploring ways to reduce overall inflammation in the body can create a healthier brain environment to support cognitive resilience.

Another sign of aging is dysregulated proteostasis, which encompasses all the processes that regulate how proteins are produced, folded and cleared within a cell. Because proteins are involved in virtually every process in the body, ensuring their proper regulation is critical for overall health. Proteostasis declines with age and is linked to age-related disorders like Alzheimer's, where misfolded amyloid and tau proteins accumulate in the brain and cannot be effectively cleared away. Understanding how age disrupts proteostasis—and uncovering ways to restore it—could extend healthy aging and reduce disease pathology.

In addition to biological factors, aging is shaped by the exposome—the cumulative impact of environmental and lifestyle factors over a lifetime. While we can't change genetic factors, as individuals and as societies, we can influence modifiable factors such as education access and diet quality. Research into the exposome will help identify ways to reduce Alzheimer's risk and promote brain health.

Defining healthy aging is only one part of understanding how to promote brain health. Another approach is the study of super-agers—individuals who remain cognitively vibrant well into their ninth and even 10th decades. These remarkable individuals often possess genetic traits that protect against dementia, offering valuable insights into how the brain can stay healthy for a lifetime. By studying super-agers, researchers hope to design interventions that help more people achieve cognitive longevity.

At Cure Alzheimer's Fund, we are committed to unlocking the secrets to lifelong brain health and identifying early disease markers. By understanding how to protect the brain as we age, we move closer to preventing Alzheimer's before it starts.

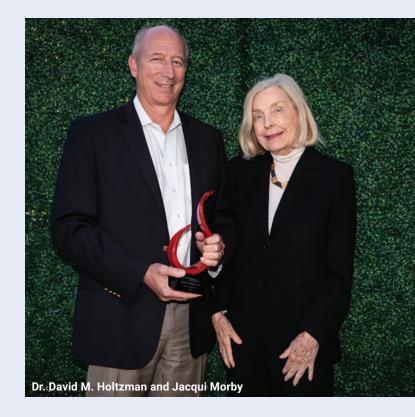
The groundbreaking research we support is drawing us nearer to an end to Alzheimer's disease. Your generosity fuels this progress, and for this, we are truly grateful.

The Inaugural Jeffrey L. Morby Prize Honors Drs. David M. Holtzman and Xiaoying Chen

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 memory, CureAlz established the annual Jeffrey L. Morby Prize. This award recognizes the senior and first authors of a recent scientific publication that significantly advances our understanding of Alzheimer's disease (AD) and opens new pathways to translate scientific results into effective ways to prevent, diagnose or treat Alzheimer's disease. Recipients of the award are determined by the community of scientists funded by CureAlz; the prize includes \$200,000 for the senior author's lab for Alzheimer's disease-related research.

At the inaugural award ceremony held on May 13, 2024, Jeff's wife and Cure Alzheimer's Fund Co-Founder Jacqui Morby delivered an inspiring speech about their shared commitment to advancing scientific research as the key to defeating this disease. Jeff's leadership encouraged both CureAlz and the broader research community to pursue deeper questions and practical insights—a spirit embodied by this year's prize recipients.

Senior author David M. Holtzman, M.D., and first author Xiaoying Chen, Ph.D., of Washington University School of Medicine in St. Louis won the award for their published paper, "Microglia-Mediated T Cell Infiltration Drives Neurodegeneration in Tauopathy" (Nature, March 2023). The study is a remarkably rich and groundbreaking exploration of the impact of agents of the peripheral adaptive immune system on neuronal loss and brain atrophy in the context of amyloid beta, tau and/or APOE4. Their work showed that in mouse models, tau pathology—but not amyloid beta—triggers the brain's immune cells (microglia) to recruit T cells from the body.



Once in the brain, these T cells interact with microglia to drive neurodegeneration. Removing either microglia or T cells prevents brain cell loss and cognitive decline. This study highlights the powerful role of the immune system—particularly the interaction between the brain and the body's immune responses—in driving disease. We hope their discoveries will pave the way to interventions targeting signaling among T cells and microglia to improve brain resilience to AD tau pathology.

Congratulations to Drs. Holtzman and Chen!

New Researchers

This gallery features researchers who received funding from Cure Alzheimer's Fund for the first time in 2024. To read about all the researchers we have supported, visit CureAlz.org/the-research/researchers/.



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



Valerija Dobricic, Ph.D. University of Lübeck, Germany



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



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



Bahaa Elgendy, Ph.D. Washington University School of Medicine in St. Louis



Michael Haney, Ph.D. University of Pennsylvania



Anil Cashikar, Ph.D. Washington University School of Medicine in St. Louis



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



Andrew J. Holbrook, Ph.D. University of California, Los Angeles



Yu Chen, Ph.D. New York University



Florent Ginhoux, Ph.D. Agency for Science, Technology and Research, Singapore



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



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



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



Theodore J. Lampidis, Ph.D. University of Miami



Cristian Lasagna-Reeves, Ph.D. Baylor College of Medicine



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



Peter Sicinski, M.D., Ph.D. Dana-Farber Cancer Institute; Harvard Medical School



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



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



Peter M. Tessier, Ph.D. University of Michigan



Diane Mathis, Ph.D. Harvard Medical School



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



Martine Therrien, Ph.D. University of California, Davis



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



Rajiv R. Ratan, M.D., Ph.D. Weill Cornell Medicine; Burke Neurological Institute



Jessica Young, Ph.D. University of Washington



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



Daniel S. Reich, M.D., Ph.D. National Institute of Neurological Disorders and Stroke, National Institutes of Health



Theodore Zwang, Ph.D. Massachusetts General Hospital; Harvard Medical School



Per Nilsson, Ph.D. Karolinska Institutet, Sweden

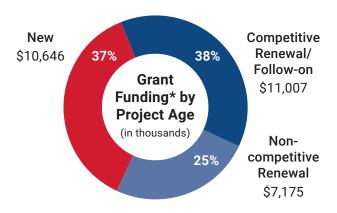


Naresha Saligrama, Ph.D. Washington University School of Medicine in St. Louis

2024 Funded Research

Cure Alzheimer's Fund spent \$30 million to support our research program that included grants for 120 research projects across four research areas of focus. To read about 2024 research projects, visit us online at **bit.ly/2024-research**.





Number of New Investigators and Projects

	Number	% of Total
New Named Investigators	32	23%
New Projects	41	34%
New Institutions	15	23%

^{*} Excludes research materials and scientific meeting spending.

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	\$219,578
Systematic Assessment of Tandem Repeats in Alzheimer's Disease (STaR-AD) Lars Bertram, M.D., University of Lübeck, Germany Valerija Dobricic, Ph.D., University of Lübeck, Germany	\$363,000
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	\$201,235
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	\$230,000
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	\$187,315
Interpreting Alzheimer's Disease-Associated Genetic Variation at Enhancer Regions Andreas R. Pfenning, Ph.D., Carnegie Mellon University	\$201,250
The Alzheimer's Genome Project™ Rudolph E. Tanzi, Ph.D., Massachusetts General Hospital; Harvard Medical School	\$1,955,000
BIOMARKERS, DIAGNOSTICS, AND STUDIES OF RISK AND RESILIENCE	
Midlife Autoantibody Profiles and the Risk of Late-Onset Alzheimer's Disease in Women Yu Chen, Ph.D., New York University	\$201,250

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	\$268,750
Utility of Blood-Based Markers for Predicting Amyloid-Related Imaging Abnormalities and Their Course	\$24,957
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	\$201,250
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	\$213,426
Understanding Human Brain Resilience to Alzheimer's Pathology Teresa Gomez-Isla, M.D., Massachusetts General Hospital; Harvard Medical School	\$300,000
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., The University of Texas Health Science Center at San Antonio Tiffany F. Kautz, Ph.D., The University of Texas Health Science Center at San Antonio	\$201,250
Bernard Fongang, Ph.D., The University of Texas Health Science Center at San Antonio	
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	\$185,140
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	\$123,098
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	\$200,388
Frances Edwards, Ph.D., University College London, England John Hardy, Ph.D., University College London, England	
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	\$201,250
Characterization and Validation of Two Recently Created Sheep Models of Alzheimer's Disease in Preparation	\$199,926
for Use as a Preclinical Pharmaceutical Testing Model Russell G. Snell, Ph.D., University of Auckland, New Zealand Natasha McKean, Ph.D., University of Auckland, New Zealand	
Genes to Therapies™ (G2T) Research Models and Materials Taconic Biosciences	\$202,715
Effect of APOE Genotype in a Novel Rat Model of Cerebral Amyloid Angiopathy William Van Nostrand, Ph.D., The University of Rhode Island	\$201,250
Dissecting Alzheimer's Disease Phenotypes in Directly Reprogrammed Patient-Derived Neurons Andrew S. Yoo, Ph.D., Washington University School of Medicine in St. Louis	\$258,750

TRANSLATIONAL RESEARCH	
STUDIES OF NOVEL ALZHEIMER'S DISEASE GENES	
Role of Ras and Rab Interactor 3 (RIN3) and Bridging Integrator 1 (BIN1) Interaction in the Neurons for Alzheimer's Disease Development Raja Bhattacharyya, Ph.D., Massachusetts General Hospital; Harvard Medical School	\$201,250
Multiomic and Functional Characterization of Soluble TREM2 Modifiers Carlos Cruchaga, Ph.D., Washington University School of Medicine in St. Louis	\$200,356
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	\$125,000
Dissecting the Modulatory Roles of Interleukin-17 Receptor D in Alzheimer's Disease Jun Huh, Ph.D., Harvard Medical School	\$201,250
Elucidating the Therapeutic Potential of the Endo-Lysosome Pathway for Alzheimer's Disease Jessica Young, Ph.D., University of Washington	\$201,250
STUDIES OF AMYLOID PRECURSOR PROTEIN AND AMYLOID BETA	
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	\$201,250
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	\$200,400
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	\$230,000
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	\$287,277
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	\$366,770
Alzheimer's Disease Tau Consortium: Toxic Consequences of Early Tau Seeding Bradley T. Hyman, M.D., Ph.D., Massachusetts General Hospital; Harvard Medical School Rachel Bennett, Ph.D., Massachusetts General Hospital; Harvard Medical School	\$287,500
Alzheimer's Disease Tau Consortium: The Role of Amyloid Beta-Induced Membrane Damage in Tau Pathology Katherine Sadleir, Ph.D., Northwestern University Feinberg School of Medicine Robert Vassar, Ph.D., Northwestern University Feinberg School of Medicine	\$286,559
Alzheimer's Disease Tau Consortium: Deep Mass Spectrometry Profiling of Tau Aggregates in Alzheimer's Disease and Other Tauopathies	\$287,500
Henrik Zetterberg, M.D., Ph.D., University of Gothenburg, Sweden; University College London, England Gunnar Brinkmalm, Ph.D., University of Gothenburg, Sweden	
Cellular Vulnerability to Pathological Tau Protein Accumulation in Alzheimer's Disease Mathieu Bourdenx, Ph.D., University College London, England Karen E. Duff, Ph.D., University College London, England	\$219,880

Multiomics Characterization of Tau Pathology Onset and its Relationship with Amyloid in the Human Hippocampus Inma Cobos, M.D., Ph.D., Stanford University	\$201,250
Identifying Mediators of Tau-Mediated Neuronal Necroptosis Using an Innovative In Vivo CRISPR Screen Bart De Strooper, M.D., Ph.D., VIB-KU Leuven, Belgium; University College London, England	\$230,000
Selective Vulnerability in Posterior Cortical Atrophy John R. Dickson, M.D., Ph.D., Massachusetts General Hospital; Harvard Medical School Bradley T. Hyman, M.D., Ph.D., Massachusetts General Hospital; Harvard Medical School	\$230,000
Tau-Induced Postsynaptic Dysfunction in Tauopathy Models Karin Hochrainer, Ph.D., Weill Cornell Medicine Costantino ladecola, M.D., Weill Cornell Medical College	\$201,250
Hypertension, Tau and Neurodegeneration Costantino ladecola, M.D., Weill Cornell Medical College Giuseppe Faraco, M.D., Ph.D., Weill Cornell Medical College	\$200,532
Modulating the Levels of Tau-Seed Interactors to Treat Alzheimer's Disease Cristian Lasagna-Reeves, Ph.D., Baylor College of Medicine	\$200,631
Investigating the Role of Tau Protein in Neuronal Senescence Miranda E. Orr, Ph.D., Washington University School of Medicine in St. Louis	\$201,250
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	\$287,500
Fleming APOE Consortium: APOE4 Accelerates CD8 Exhaustion via Glucocorticoid Signaling in Alzheimer's Female Carriers Oleg Butovsky, Ph.D., Brigham and Women's Hospital; Harvard Medical School Vijay K. Kuchroo, D.V.M., Ph.D., Brigham and Women's Hospital; Harvard Medical School	\$575,000
Fleming APOE Consortium: Cell Autonomous Roles of Protective APOE Variants in Microglia in Response to Amyloid Pathology Michael Haney, Ph.D., University of Pennsylvania	\$285,952
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	\$345,000
Fleming APOE Consortium: Effect of Cholesteryl Ester Transfer Protein Activity on Amyloid and Cerebrovascular Pathologies in Animal Models of Alzheimer's Disease Cheryl Wellington, Ph.D., University of British Columbia, Canada	\$287,500
Investigating Lysosomal Mechanisms of Risk and Resilience in Alzheimer's Disease Joel Blanchard, Ph.D., Icahn School of Medicine at Mount Sinai	\$201,250
APOE in Choroid Plexus Function and Related Alzheimer's Disease Pathogenesis Guojun Bu, Ph.D., The Hong Kong University of Science and Technology	\$230,000
Circuit Dynamics in APOE4 Mice Ksenia Kastanenka, Ph.D., Massachusetts General Hospital; Harvard Medical School	\$201,227
Neuroproteasomes Mechanistically Connect APOE Isoforms to Endogenous Tau Aggregation Kapil V. Ramachandran, Ph.D., Columbia University	\$201,279

2024 FUNDED RESEARCH (CONTINUED)

STUDIES OF THE IMMUNE RESPONSE IN ALZHEIMER'S DISEASE	
Neuroimmune Consortium: Astrocyte Inflammatory Contributions to Alzheimer's Disease Shane A. Liddelow, Ph.D., New York University	\$287,500
Neuroimmune Consortium: Impact of AD Polygenic Risk Score on Microglial Response to Peripheral Inflammation Martine Therrien, Ph.D., University of California, Davis	\$285,851
2024 Jeffrey L. Morby Prize for Exceptional Research David M. Holtzman, M.D., Washington University School of Medicine in St. Louis	\$201,250
The Role of Astrocyte-Secreted Insulin-Like Growth Factor Binding Protein 2 (IGFBP2) in the Progression of Alzheimer's Disease Nicola Allen, Ph.D., Salk Institute for Biological Studies	\$201,250
Contributions of IL34 Signaling to Microglial Function and Alzheimer's Pathology in Mice Staci Bilbo, Ph.D., Duke University School of Medicine	\$192,314
Defining a Role for the MS4A Genes in Alzheimer's Disease Sandeep Robert Datta, M.D., Ph.D., Harvard Medical School	\$201,250
VGF-Derived Peptide Therapy for Alzheimer's Disease: Studies of Mouse and Human TLQP-21 and its Receptor, C3aR1 Michelle E. Ehrlich, M.D., Icahn School of Medicine at Mount Sinai Stephen R. Salton, M.D., Ph.D., Icahn School of Medicine at Mount Sinai	\$172,500
Tau and Amyloid Beta are Innate Immune Antimicrobial Peptides in the Brain William Eimer, Ph.D., Massachusetts General Hospital; Harvard Medical School	\$201,250
Targeting Reactive Astrocytes AMPK Signaling to Suppress Inflammation in Alzheimer's Disease Gilbert Gallardo, Ph.D., Washington University School of Medicine in St. Louis	\$201,250
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	\$201,250
Multidimensional Profiling of TREM2-Mutated or APOE4-Mutated Microglia in Human Brain Organoids to Understand Dysregulated Microglia Neuronal Crosstalk in Alzheimer's Disease Florent Ginhoux, Ph.D., Agency for Science, Technology and Research, Singapore	\$201,250
Endogenous Human Antibodies Associated with Alzheimer's Disease Charles Glabe, Ph.D., University of California, Irvine	\$230,000
Investigating Alzheimer's Disease-Associated Membrane Biology in Microglia and Neurons Anna Greka, M.D., Ph.D., Brigham and Women's Hospital; Harvard Medical School; Broad Institute Beth Stevens, Ph.D., Boston Children's Hospital; Harvard Medical School; Broad Institute	\$230,000
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	\$201,250
Role of CD8+ T Cell-Glial Interactions in Mediating Alzheimer's Disease Pathogenesis Mehdi Jorfi, Ph.D., Massachusetts General Hospital; Harvard Medical School Joseph Park, Ph.D., Massachusetts General Hospital; Harvard Medical School Rudolph E. Tanzi, Ph.D., Massachusetts General Hospital; Harvard Medical School	\$201,250
To Elucidate the Role of Memory T Cells as a Determinant of Age-Related Inflammation in Alzheimer's Disease Susan Kaech, Ph.D., Salk Institute for Biological Studies	\$201,250

Contribution of Skull Bone Marrow-Derived Cells to Alzheimer's Disease Jonathan Kipnis, Ph.D., Washington University School of Medicine in St. Louis	\$201,250
Role of Checkpoint Molecules TIM-3 and LAG-3 in Microglial Function in Alzheimer's Disease Vijay K. Kuchroo, D.V.M., Ph.D., Brigham and Women's Hospital; Harvard Medical School	\$201,250
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., Brigham and Women's Hospital; Harvard Medical School	\$228,779
The Role of Interferon-Induced Transmembrane Protein 3 (IFITM3) and Gamma-Secretase in Microglia Yueming Li, Ph.D., Memorial Sloan Kettering Cancer Center	\$230,000
Elucidating the Role of CLEC7A in Tau-Mediated Neurodegenerative Disease John R. Lukens, Ph.D., University of Virginia	\$201,250
Meningeal Regulatory T Cells (Tregs) in Individuals With Versus Without Alzheimer's Disease Diane Mathis, Ph.D., Harvard Medical School	\$201,250
Investigating the Association Between Clonal Hematopoiesis and Alzheimer's Disease Cameron McAlpine, Ph.D., Icahn School of Medicine at Mount Sinai	\$201,250
Elucidating Mechanisms Driving the Compromised Balance Between Mitophagy and cGAS-STING-Initiated Inflammation Toward a Treatment for Alzheimer's Disease Per Nilsson, Ph.D., Karolinska Institutet, Sweden Evandro F. Fang, Ph.D., University of Oslo, Akershus University Hospital, Norway	\$198,490
Probing the Molecular Underpinnings of G Protein-Coupled Receptor ADGRG1 Mediated Protective Microglial Responses to Alzheimer's Disease Xianhua Piao, M.D., Ph.D., University of California, San Francisco	\$201,250
Extracellular ATP is a Key Factor in Promoting Alzheimer's Disease Neuroinflammation Paola Pizzo, Ph.D., University of Padova, Italy Anna Lisa Giuliani, Ph.D., University of Ferrara, Italy	\$149,995
Specificity of T-Cell Responses in Autosomal Dominant Alzheimer's Disease (ADAD) Naresha Saligrama, Ph.D., Washington University School of Medicine in St. Louis	\$201,250
Dissecting Microglial State Dynamics in Alzheimer's Disease Li-Huei Tsai, Ph.D., Massachusetts Institute of Technology; Broad Institute	\$300,000
T-Cell Modulation of Microglia to Treat Alzheimer's Disease Howard L. Weiner, M.D., Brigham and Women's Hospital; Harvard Medical School	\$201,234
Decipher the Astrocyte Cell-Surface Proteome in Alzheimer's Disease Hui Zheng, Ph.D., Baylor College of Medicine Junmin Peng, Ph.D., St. Jude Children's Research Hospital	\$230,000
STUDIES OF ALTERNATIVE NEURODEGENERATIVE PATHWAYS	
Brain Exit and Entry Consortium: Human Three-Dimensional Neuro-Vascular Interaction and Meningeal Lymphatics Models with Application to Alzheimer's Disease Se Hoon Choi, Ph.D., Massachusetts General Hospital; Harvard Medical School	\$287,500
Brain Entry and Exit Consortium: How Does Vascular Fatty Acid Metabolism Regulate the Pathophysiology of Alzheimer's Disease?	\$287,500
Richard Daneman, Ph.D., University of California, San Diego	

2024 FUNDED RESEARCH (CONTINUED)

Project/Researcher	Distribution Amount
Project/Researcher	DISTIDUTION ATTIONIT

Brain Entry and Exit Consortium: Meningeal Mast Cell Control of Cerebrospinal Fluid Dynamics in Homeostasis and Alzheimer's Disease	\$345,000
Jonathan Kipnis, Ph.D., Washington University School of Medicine in St. Louis	
Brain Entry and Exit Consortium: Neuroinflammation at the Choroid Plexus in Alzheimer's Disease Maria K. Lehtinen, Ph.D., Boston Children's Hospital; Harvard Medical School	\$86,250
Brain Entry and Exit Consortium: Does Subarachnoid Lymphatic-Like Membrane (SLYM) Failure Compromise Glymphatic Clearance in Alzheimer's Disease?	\$287,500
Maiken Nedergaard, M.D., D.M.Sc., University of Rochester; University of Copenhagen, Denmark	
Brain Entry and Exit Consortium: High-Resolution Magnetic Resonance Imaging of the Brain Borders Daniel S. Reich, M.D., Ph.D., National Institute of Neurological Disorders and Stroke, National Institutes of Health	\$287,500
Microbiome Consortium: Harnessing Diet-Microbe Interactions to Prevent Alzheimer's Disease Pathogenesis Laura M. Cox, Ph.D., Brigham and Women's Hospital; Harvard Medical School	\$275,500
Microbiome Consortium: The Role of Gut Microbial Metabolism in Tau-Mediated Neurodegeneration David M. Holtzman, M.D., Washington University School of Medicine in St. Louis	\$287,500
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	\$250,000
Microbiome Consortium: Microbial Profiling of Human Brain and Gut Microbiomes in Alzheimer's Disease Rudolph E. Tanzi, Ph.D., Massachusetts General Hospital; Harvard Medical School Nanda Kumar Navalpur Shanmugam, Ph.D., Massachusetts General Hospital; Harvard Medical School	\$287,500
Microbiome Consortium: Interaction of the Microbiome with Astrocytes and Amyloid Pathology Robert Vassar, Ph.D., Northwestern University Feinberg School of Medicine	\$345,000
Identifying Age-Related Proteomic Changes That Predict Future Onset of Amyloid Beta Aggregation in Late-Onset Alzheimer's Disease Randall J. Bateman, M.D., Washington University School of Medicine in St. Louis	\$201,250
Neuroprotective Effects of the Exercise Hormone Irisin in Alzheimer's Disease Se Hoon Choi, Ph.D., Massachusetts General Hospital; Harvard Medical School Christiane Wrann, D.V.M., Ph.D., Massachusetts General Hospital; Harvard Medical School	\$345,000
Restore Meningeal Lymphatic Drainage to Alleviate White Matter Damage and Cerebral Amyloid Angiopathy in a Model of Alzheimer's Disease Sandro Da Mesquita, Ph.D., Mayo Clinic, Jacksonville	\$201,250
Decoding Microbial Products Modulating Alzheimer's Disease—Toward Precision Postbiotics Treatment Eran Elinav, M.D., Ph.D., Weizmann Institute of Science, Israel; DKFZ, Germany	\$201,250
Turning Up Mitophagy to Blunt Alzheimer's Tau Pathologies Evandro F. Fang, Ph.D., University of Oslo, Akershus University Hospital, Norway	\$200,675
The Role of Calcium Homeostasis in Axonal Spheroid Formation in Alzheimer's Disease Jaime Grutzendler, M.D., Yale School of Medicine	\$199,995
Novel Artificial Intelligence (AI) Decodes Aging Neurons Andrew J. Holbrook, Ph.D., University of California, Los Angeles Theodore Zwang, Ph.D., Massachusetts General Hospital; Harvard Medical School	\$201,250

Characterizing Gut Bacteriome-Mycobiome Synergy in Correlation to Amylin-Amyloid Beta Antimicrobial Synergy in Alzheimer's Disease (AD) in AD Mouse Models	\$201,250
Deepak Kumar Vijaya Kumar, Ph.D., Massachusetts General Hospital; Harvard Medical School	
Dysregulation of Signaling on Post Synaptic Density Scaffolds in Alzheimer's Disease Alexandra C. Newton, Ph.D., University of California, San Diego	\$201,250
Evaluating the Contribution of TDP-43 Dysfunction and Cryptic Mis-Splicing to Alzheimer's Disease Pathogenesis Leonard Petrucelli, Ph.D., Mayo Clinic, Jacksonville	\$201,250
Protection Against Alzheimer's Disease with Longevity-Promoting Intervention 17a-Estradiol Christian Pike, Ph.D., University of Southern California Bérénice A. Benayoun, Ph.D., University of Southern California	\$227,410
2-Deoxyglucose and Its Analogs as Novel Therapeutics to Build Resilience to Alzheimer's Disease Rajiv R. Ratan, M.D., Ph.D., Weill Cornell Medicine; Burke Neurological Institute Theodore J. Lampidis, Ph.D., University of Miami	\$197,596
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., Dana-Farber Cancer Institute; Harvard Medical School	\$201,250
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	\$216,287
Role of Psychosocial Stress in Alzheimer's Disease Filip Swirski, Ph.D., Icahn School of Medicine at Mount Sinai	\$201,250
Role of the Circulating Exerkine GPLD1 in Ameliorating Alzheimer's Disease Pathology Saul Villeda, Ph.D., University of California, San Francisco	\$201,250
Understanding How Human Brain Vascular Cells Mediate Genetic Risk for Alzheimer's Disease Andrew Yang, Ph.D., University of California, San Francisco	\$201,250
DRUG DISCOVERY	
DRUG SCREENING AND LEAD DRUG EVALUATION PROJECTS	
Development of Small Molecule Inhibitors of Cholesterol 25-hydroxylase Anil Cashikar, Ph.D., Washington University School of Medicine in St. Louis Bahaa Elgendy, Ph.D., Washington University School of Medicine in St. Louis	\$201,250
Validation and Characterization of Compounds Modulating Neuroinflammation and Amyloid Beta Uptake in Microglial Cells Ana Griciuc, Ph.D., Massachusetts General Hospital; Harvard Medical School Luisa Quinti, Ph.D., Massachusetts General Hospital; Harvard Medical School	\$201,250
Exploring Novel Drug Candidates for Alzheimer's Disease Through Integrative Pathway Analysis and Validation in 3D Cellular Models Doo Yeon Kim, Ph.D., Massachusetts General Hospital; Harvard Medical School Luisa Quinti, Ph.D., Massachusetts General Hospital; Harvard Medical School	\$201,250
Identification and Development of CD33 Inhibitors and Pre-RNA Splicing Modulators Subhash Sinha, Ph.D., Weill Cornell Medicine	\$201,250

2024 FUNDED RESEARCH (CONTINUED)

PRECLINICAL AND CLINICAL DRUG DEVELOPMENT	
PRECLINICAL DRUG DEVELOPMENT	
Combined Hormone Therapy as a Novel Treatment for Alzheimer's Disease in the Face of a Metabolic Challenge: Influence of Sex and Genotype	\$201,250
Liisa Galea, Ph.D., University of British Columbia, Canada; Centre for Addiction and Mental Health, Canada Annie Ciernia, Ph.D., University of British Columbia, Canada	
Sex-Biased Toll-Like Receptor 7 (TLR7) Signaling in Demyelination and Its Inhibition by Small Molecules Li Gan, Ph.D., Weill Cornell Medicine Subhash Sinha, Ph.D., Weill Cornell Medicine	\$402,500
Targeting Neuroinflammation with Nasal Administration of Anti-CD3 Monoclonal Antibody to Treat Alzheimer's Disease Rafael M. Rezende, Ph.D., Brigham and Women's Hospital; Harvard Medical School	\$195,250
Non-Invasive Delivery of IL-2 to the CNS for Local Expansion of Regulatory T Cells and Prevention of Neurodegeneration in Tauopathy	\$201,250
Peter M. Tessier, Ph.D., University of Michigan David M. Holtzman, M.D., Washington University School of Medicine in St. Louis	
Preclinical Analysis of Synaptogyrin-3 Oligonucleotides to Target Tauopathy Patrik Verstreken, Ph.D., VIB-KU Leuven, Belgium	\$201,250
Characterization of CNS-Penetrant HDAC11 Selective Inhibitors in Alzheimer's Disease Models Can (Martin) Zhang, M.D., Ph.D., Massachusetts General Hospital; Harvard Medical School Changning Wang, Ph.D., Massachusetts General Hospital; Harvard Medical School	\$201,250
CLINICAL TRIALS	
A Proposal to Evaluate the Effect of Bacillus Calmette-Guérin Vaccination on Alzheimer's Disease Development Tamir Ben-Hur, M.D., Ph.D., Hadassah University Medical Center, Israel Herve Bercovier, D.V.M., M.Sc., Hebrew University of Jerusalem, Israel	\$121,900
OTHER	
SCIENTIFIC MEETINGS AND SUPPORT	
Scientific Meeting Support	\$281,717

Ongoing Research Projects

The research projects listed here were ongoing and active in 2024, having received funding from Cure Alzheimer's Fund in a previous year.

Figet/Nesearcher Distr	ibution Amount
FOUNDATIONAL RESEARCH	
BIOLOGICAL RESEARCH MATERIALS: NEW ANIMAL AND CELLULAR MODELS, AND HUMAN SAMPLES	
Adding Genomics and Methylomics to Personalized Disease Prediction for Alzheimer's Disease (EPIC4AD) Lars Bertram, M.D., University of Lübeck, Germany Christina M. Lill, M.D., M.Sc., University of Münster, Germany; Imperial College London, England	\$116,684
Influence of Plaque Vicinity on Microglial and Astrocyte Gene Expression; Role of Human Tau and TREM2 Frances Edwards, Ph.D., University College London, England John Hardy, Ph.D., University College London, England	\$172,369
Cerebrospinal Fluid Neuroinflammatory Signature in Alzheimer's Disease and Related Proteopathies Mathias Jucker, Ph.D., German Center for Neurodegenerative Diseases (DZNE); University of Tübingen, Germany Stephan Kaeser, Ph.D., University of Tübingen, Germany Stefan Lichtenthaler, Ph.D., German Center for Neurodegenerative Diseases (DZNE); Technische Universität München (TUM), Germany	\$180,550
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	\$541,897
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	\$144,325
Neurobiological Basis of Cognitive Impairment in African Americans: Deep Phenotyping of Older African Americans at Risk of Dementia—The Dementia (in) African American Population Phenotyping (for) Potential Elevated Risk (DAAPPER) Study Henry L. Paulson, M.D., Ph.D., University of Michigan Bruno Giordani, Ph.D., University of Michigan Benjamin M. Hampstead, Ph.D., ABPP/CN, University of Michigan	\$243,407
Multidisciplinary Phenotyping of a Novel Humanized LOAD Mouse Model Giuseppina Tesco, M.D., Ph.D., Tufts University School of Medicine	\$201,250
EPIGENETIC FACTORS	
CIRCUITS: Characterizing Epigenetic Biomarkers of Human Cognitive Aging Lars Bertram, M.D., University of Lübeck, Germany	\$252,250
CIRCUITS: A Unified Approach to Actionable Alzheimer's Disease Signatures Winston Hide, Ph.D., Beth Israel Deaconess Medical Center; Harvard Medical School	\$248,980
TRANSLATIONAL RESEARCH	
STUDIES OF NOVEL ALZHEIMER'S DISEASE GENES	
Exploring the Therapeutic Potential of Clusterin in a Preclinical Model of Alzheimer's Disease Alban Gaultier, Ph.D., University of Virginia	\$201,250
ABCA7 Loss of Function in Aging and Alzheimer's Disease Takahisa Kanekiyo, M.D., Ph.D., Mayo Clinic, Jacksonville	\$201,250

ONGOING RESEARCH PROJECTS (CONTINUED)

r toject/ Nesearcher Distrik	Julion Amount
STUDIES OF AMYLOID PRECURSOR PROTEIN AND AMYLOID BETA	
SFRP1 as a Therapeutic Target and Diagnostic/Prognostic Factor in Alzheimer's Disease Paola Bovolenta, Ph.D., Universidad Autónoma de Madrid, Spain	\$172,500
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	\$230,000
STUDIES OF TAU	
Alzheimer's Disease Tau Consortium: Role of VCP/p97 in Tau Prion Replication Marc I. Diamond, M.D., University of Texas Southwestern Medical Center	\$287,000
Alzheimer's Disease Tau Consortium: Impact of Tau Mutations and Amyloid Beta on Tau Post-Translational Modifications and Conformation Karen E. Duff, Ph.D., University College London, England	\$344,993
Alzheimer's Disease Tau Consortium: How Do Soluble Tau Species Replicate? Bradley T. Hyman, M.D., Ph.D., Massachusetts General Hospital; Harvard Medical School	\$287,169
Toxic Effects of Extracellular Tau Oligomers on Neurons George S. Bloom, Ph.D., University of Virginia	\$192,020
RNA as a Determinant of Tau Seeding Marc I. Diamond, M.D., University of Texas Southwestern Medical Center	\$230,000
Characterization of Tau Pathology Heterogeneity Across the Alzheimer's Disease Spectrum Oskar Hansson, M.D., Ph.D., Lund University, Sweden Rik Ossenkoppele, Ph.D., Amsterdam University Medical Center, The Netherlands; Lund University, Sweden	\$201,250
Using Long-Read Sequencing to Investigate the MAPT Locus and Transcripts in Neurodegeneration John Hardy, Ph.D., University College London, England	\$201,250
Regional Variability of Pathology-Associated Properties of Tau in Posterior Cortical Atrophy Bradley T. Hyman, M.D., Ph.D., Massachusetts General Hospital; Harvard Medical School John R. Dickson, M.D., Ph.D., Massachusetts General Hospital; Harvard Medical School	\$172,000
Targeting Microglial TSG101 for Synaptic Protection and Cognitive Enhancement in Alzheimer's Disease Seiko Ikezu, M.D., Mayo Clinic, Jacksonville Tsuneya Ikezu, M.D., Ph.D., Mayo Clinic, Jacksonville	\$172,500
STUDIES OF APOLIPOPROTEIN E (APOE)	
Fleming APOE Consortium: APOE4-Mediated Dysfunction of CD8 T Cell–Microglia Crosstalk in Alzheimer's Disease Oleg Butovsky, Ph.D., Brigham and Women's Hospital; Harvard Medical School	\$287,500
Fleming APOE Consortium: Role of APOE Isoforms in Immune Responses in a Model of Tauopathy David M. Holtzman, M.D., Washington University School of Medicine in St. Louis	\$345,000
Fleming APOE Consortium: Modulation of Selective Neuronal Vulnerability in Alzheimer's Disease by Apolipoprotein E Jean-Pierre Roussarie, Ph.D., Boston University	\$287,500
Mitochondrial Alzheimer's Risk Factors Control APOE Expression and Secretion Victor Faundez, M.D., Ph.D., Emory University	\$201,250
Sex Matters: Understanding the Influence of Sex and Apolipoprotein E (APOE) Genotype on Hippocampal Plasticity and Cognition Liisa Galea, Ph.D., University of British Columbia, Canada; Centre for Addiction and Mental Health, Canada	\$170,200
Elucidating the Protective Effects of APOE2 in the Presence of APOE4 Gene Allele in Animal Models Na Zhao, M.D., Ph.D., Mayo Clinic, Jacksonville Yingxue Ren, Ph.D., Mayo Clinic, Jacksonville	\$201,250

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	\$287,493
Neuroimmune Consortium: Mechanisms Mediating Microglia Sensing of Peripheral Inflammation Christopher K. Glass, M.D., Ph.D., University of California, San Diego	\$287,500
Neuroimmune Consortium: Biomarker Tool Development Jacob Hooker, Ph.D., Massachusetts General Hospital; Harvard Medical School	\$287,500
Neuroimmune Consortium: Effects of Peripheral Inflammation on Myeloid Cell Function in Alzheimer's Disease Beth Stevens, Ph.D., Boston Children's Hospital; Harvard Medical School; Broad Institute	\$344,085
Prenatal Inflammation Effects on Blood-Brain Barrier Function and Alzheimer's Disease-Related Pathologies Across the Lifespan Alexandre Bonnin, Ph.D., Children's Hospital Los Angeles; University of Southern California	\$201,250
A New Model of Microglia Genetic Perturbation in Vivo to Screen All Risk Factors Associated with Alzheimer's Disease Oleg Butovsky, Ph.D., Brigham and Women's Hospital; Harvard Medical School Vijay K. Kuchroo, D.V.M., Ph.D., Brigham and Women's Hospital; Harvard Medical School	e \$431,250
Microglial-Specific INPP5D Knockdown Modulates Behavior, Amyloidosis and Tauopathy in Alzheimer's Mouse Models Samuel E. Gandy, M.D., Ph.D., Icahn School of Medicine at Mount Sinai Michelle E. Ehrlich, M.D., Icahn School of Medicine at Mount Sinai	\$217,327
T-Cell Epigenetics in Alzheimer's Disease David M. Gate, Ph.D., Northwestern University Feinberg School of Medicine	\$172,500
Investigating MEF2C Transcription Factor as a Therapeutic Target to Reprogram Pathological Microglial States in Alzheimer's Disease Alison M. Goate, D.Phil., Icahn School of Medicine at Mount Sinai Edoardo Marcora, Ph.D., Icahn School of Medicine at Mount Sinai	\$201,250
Revealing New Genes and Pathways at the Intersection of Lipotoxic and Genetic Risk for Alzheimer's Disease Anna Greka, M.D., Ph.D., Brigham and Women's Hospital; Harvard Medical School; Broad Institute Beth Stevens, Ph.D., Boston Children's Hospital; Harvard Medical School; Broad Institute	\$172,550
Signaling Function of TREM2 Cleavage Products, Which Are Affected by Agonistic Antibodies to the Stalk Region Christian Haass, Ph.D., German Center for Neurodegenerative Diseases (DZNE), Germany Kai Schlepckow, Ph.D., German Center for Neurodegenerative Diseases (DZNE), Germany	\$172,500
Mechanisms of Astrocyte-Derived Lipid Toxicity in Alzheimer's Disease Shane A. Liddelow, Ph.D., New York University	\$208,033
Neuroimmune Connectome Perturbations in Alzheimer's Disease Francisco J. Quintana, Ph.D., Brigham and Women's Hospital; Harvard Medical School; Broad Institute	\$201,250
Human Brain CD33 Ligand, Receptor Protein Tyrosine Phosphatase Zeta (RPTPZ)S3L, Limits Microglial Phagocytosis and Contributes to Alzheimer's Disease Progression Ronald L. Schnaar, Ph.D., The Johns Hopkins University Philip C. Wong, Ph.D., The Johns Hopkins University	\$201,250
Understanding the Dynamic Lipid-Immunometabolome of Protective and Risk Alzheimer's Microglia Rik van der Kant, Ph.D., Amsterdam University Medical Center, The Netherlands	\$201,250
Elucidating the Role of Soluble Epoxide Hydrolase and Arachidonic Acid Metabolism in Neuroinflammation and Alzheimer's Disease Hui Zheng, Ph.D., Baylor College of Medicine	\$167,637

ONGOING RESEARCH PROJECTS (CONTINUED)

· ·	Jtion Amount
STUDIES OF ALTERNATIVE NEURODEGENERATIVE PATHWAYS	
Brain Entry and 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., State University of New York at Stony Brook	\$204,081
Brain Entry and Exit Consortium: A 3D In Vitro Neurovascular Human Brain Model with Meningeal Lymphatics for Elucidating Mechanisms Underlying Alzheimer's Disease Se Hoon Choi, Ph.D., Massachusetts General Hospital; Harvard Medical School	\$230,000
Brain Entry and Exit Consortium: Identifying the Blood-Brain Barrier Changes During Alzheimer's Disease Richard Daneman, Ph.D., University of California, San Diego	\$287,500
Brain Entry and Exit Consortium: Crosstalk of Central Nervous System Barriers and Clearance Routes in Homeostasis and Alzheimer's Disease Jonathan Kipnis, Ph.D., Washington University School of Medicine in St. Louis	\$345,000
Brain Entry and Exit Consortium: Biochemical and Functional Analysis of Cerebrospinal Fluid and Lymph Following Changes in Brain Fluid Dynamics Laura Santambrogio, M.D., Ph.D., Weill Cornell Medicine	\$287,500
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., University of Connecticut Health Center Riqiang Yan, Ph.D., University of Connecticut Health Center	\$230,000
Disentangling the Role of Intracranial Arteriosclerosis in Alzheimer's Disease Daniel Bos, M.D., Ph.D., Erasmus University Medical Center, The Netherlands Meike Vernooij, M.D., Ph.D., Erasmus University Medical Center, The Netherlands Frank J. Wolters, M.D., Ph.D., Erasmus University Medical Center, The Netherlands Geert Jan Biessels, M.D., Ph.D., University Medical Center Utrecht, The Netherlands Julia Neitzel, Ph.D., Harvard T.H. Chan School of Public Health	\$167,207
Scaling the Divide in Alzheimer's Disease: An Integrated Molecular, Cellular and Network-Level Study Marc Aurel Busche, M.D., Ph.D., University College London, England Samuel Harris, Ph.D., University College London, England	\$191,624
Air Pollution and Alzheimer's Disease Risk Interact with Premature Aging of Neural Stem Cells and Apolipoprotein E Alleles Caleb E. Finch, Ph.D., University of Southern California Michael A. Bonaguidi, Ph.D., University of Southern California	\$301,069
In Vivo Models for Golgi Fragmentation and the Molecular Pathogenesis of Alzheimer's Disease Samuel E. Gandy, M.D., Ph.D., Icahn School of Medicine at Mount Sinai	\$230,000
Oligodendroglial Dynamics and Myelination in Alzheimer's Disease Erin M. Gibson, Ph.D., Stanford University	\$198,751
Understanding the Mechanism Underlying Vaccination for Alzheimer's Disease Charles L. Greenblatt, M.D., Hebrew University of Jerusalem, Israel Ofer N. Gofrit, M.D., Ph.D., Hebrew University of Jerusalem, Israel Benjamin Y. Klein, M.D., Hebrew University of Jerusalem, Israel	\$115,805
Deciphering and Restoring Computational Setpoints in Alzheimer's Disease Through Sleep-Enhanced Network Homeostasis Keith B. Hengen, Ph.D., Washington University in St. Louis	\$189,902
Morphological, Electrophysiological and Transcriptional Characterization of Single Neurons from Resilient and Susceptible Models of Human Alzheimer's Disease Catherine Kaczorowski, Ph.D., University of Michigan Shannon Moore, Ph.D., University of Michigan	\$201,250
Neuronal Mechanisms Driving Synapse Loss in Alzheimer's Disease Martin Kampmann, Ph.D., University of California, San Francisco	\$201,250
Circadian Perturbations of the Vasculome and Microgliome in Alzheimer's Disease	\$200,417

Circadian Desynchrony, Glial Dysfunction and Alzheimer's Disease Pathogenesis Erik S. Musiek, M.D., Ph.D., Washington University School of Medicine in St. Louis	\$198,994
Evaluating TMEM106B Accumulation in Alzheimer's Disease Leonard Petrucelli, Ph.D., Mayo Clinic, Jacksonville Casey N. Cook, Ph.D., Mayo Clinic, Jacksonville	\$201,250
Stress and Neurovascular-Immune Networks in Alzheimer's Disease Scott J. Russo, Ph.D., Icahn School of Medicine at Mount Sinai Wolfram C. Poller, M.D., Icahn School of Medicine at Mount Sinai	\$172,500
Functional Changes to Cerebrospinal Fluid Immune Cells Resulting from Bacillus Calmette-Guérin (BCG) Vaccination in Older Adults With and Without Alzheimer's Disease	\$258,750
Marc Weinberg, M.D., Ph.D., Massachusetts General Hospital; Harvard Medical School Steven E. Arnold, M.D., Massachusetts General Hospital; Harvard Medical School	
Targeting the Microbiome and Innate Immunity in Alzheimer's Disease Howard L. Weiner, M.D., Brigham and Women's Hospital; Harvard Medical School Laura M. Cox, Ph.D., Brigham and Women's Hospital; Harvard Medical School	\$201,250
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., Houston Methodist Research Institute; Weill Cornell Medicine	\$201,199
Noncoding Translation Feedback Loop in Alzheimer's Disease Xuebing Wu, Ph.D., Columbia University	\$201,250
DRUG DISCOVERY	
DRUG SCREENING AND LEAD DRUG EVALUATION PROJECTS	
Alzheimer's Disease Drug Discovery and Development Consortium: High-Throughput Drug Screening for Alzheimer's Disease Using Three-Dimensional Human Neural Culture Systems Doo Yeon Kim, Ph.D., Massachusetts General Hospital; Harvard Medical School Luisa Quinti, Ph.D., Massachusetts General Hospital; Harvard Medical School	\$230,590
Alzheimer's Disease Drug Discovery and Development Consortium: Blocking Synaptotoxicity in Alzheimer's Three-Dimensional Models Weiming Xia, Ph.D., Boston University	\$197,500
PRECLINICAL AND CLINICAL DRUG DEVELOPMENT	
PRECLINICAL DRUG DEVELOPMENT	
Development of Human cGAS Inhibitors to Treat Alzheimer's Disease Li Gan, Ph.D., Weill Cornell Medicine Subhash Sinha, Ph.D., Weill Cornell Medicine	\$250,000
Interrogating Levetiracetam's Impact on Amyloid Pathology and Presynaptic Proteostasis in Knock-In Mouse Models with Humanized Amyloid Beta Jeffrey Savas, Ph.D., Northwestern University Feinberg School of Medicine	\$136,827
CLINICAL TRIAL DESIGN	
Application of Machine Learning Methods in Alzheimer's Disease Clinical Trials Ali Ezzati, M.D., University of California, Irvine Richard B. Lipton, M.D., Albert Einstein College of Medicine	\$100,000
OTHER	
SCIENTIFIC MEETINGS AND SUPPORT	
General Scientific Support Wilma Wasco, Ph.D., Massachusetts General Hospital; Harvard Medical School	\$95,594

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Working Together Toward a Cure

Alzheimer's disease affects millions, but it also unites a powerful community determined to find a cure. Each person's experience is unique, yet together, we are making a difference—raising funds, awareness and hope. By supporting research in ways that reflect their passions and abilities, our donors are helping to drive real progress toward a future without Alzheimer's.

Together, we can move research forward and bring hope to millions.

Cure Alzheimer's Fund supporters are an active and engaged community working toward the shared goal of finding a cure for Alzheimer's disease. Whether gathering for in-person or virtual events, rallying friends and family or designing unique fundraisers, each member of our community plays a crucial role in advancing research.

Across the country, our supporters have found creative ways to contribute—hosting dinners and cocktail receptions, organizing road races and golf tournaments, and even leading a hunt for morel mushrooms. These events do more than raise funds; they unite people in their commitment to end Alzheimer's.

Every action, no matter the size, builds momentum. By coming together, our community is supporting the breakthroughs needed to prevent, slow and ultimately cure Alzheimer's disease. Through their generosity and dedication, our supporters are proving that collective action leads to lasting impact.

Learn more about how you can be part of the movement to end Alzheimer's.

Info@CureAlz.org 781-237-3800

BETSY WEINGARTEN

"My dad, Murray Weingarten, lit up a room with his smile and laughter," says Betsy Weingarten. "He made those around him feel respected and valued. He had great enthusiasm and was passionate about everything he did. He grew up in the Bronx as the son of immigrant parents and served in the Navy during World War II. He worked hard and built a successful career, retiring as CEO of an aerospace company. He was a leader in service to those in his community. He played, watched and loved sports. He shared a loving marriage of over 61 years with my mom, Shirley, and supported my brother, me and his two grandsons in everything we ever did. Losing this smart, vibrant, loving man piece by piece to dementia was a pain I never could have imagined. I support the Cure Alzheimer's Fund so that someday, no other families will have to experience this type of loss."



THE BRASCH AND SHAW FAMILIES

Two California couples, Alicia and Sam Brasch, and Kate and Mark Shaw, have harnessed the power of community to support groundbreaking Alzheimer's research. Both families have transformed their personal experiences with Alzheimer's into action, bringing friends and neighbors together in the fight against the disease.

Sam and Mark met at Stanford in 1992, and their friendship grew after they married and had children. Alicia, Sam, Kate and Mark also strengthened their bonds through the shared experience of losing parents and other loved ones to Alzheimer's disease.

In March 2021, amid the challenges of the pandemic, the Brasches and Shaws co-hosted a virtual event to raise awareness and funds for Cure Alzheimer's Fund. They furthered their commitment last October when they organized an intimate dinner in Mill Valley, gathering members of their community from across the Bay Area. The event raised more than \$65,000—all of which will go directly to research aimed at preventing, slowing and ultimately curing Alzheimer's.

"Being that Familial Alzheimer's Disease (FAD) runs in my family, I wholeheartedly align myself with organizations like CureAlz that

From left: Sam Brasch, Mark Shaw, Kate Shaw and Alicia Brasch.

can move the needle in finding innovative cures for FAD, which inevitably will have an impact on the broader Alzheimer's field," says Alicia Brasch, Co-Founder of Youngtimers, an organization to help those affected by FAD.

Through their leadership, the Brasch and Shaw families have demonstrated the power of collective action. By lending their voices, connections and dedication to CureAlz, they are making a meaningful difference in the search for a cure.

GLENN CAFFERY — ALZRUN

"Alzheimer's hit our family hard, afflicting my otherwise healthy dad in his mid-50s, and subsequently his brother and sister," says Glenn Caffery. "Crushed by my dad's experience, and in honor of my mom's care for him through devastating symptoms and her own grief, I set out on a transcontinental fundraising run for CureAlz in 2011.

"CureAlz is an international force to end Alzheimer's, yet feels like family. They are brilliant, lovely people who make contributions from people like me matter. I did not anticipate supporting them would change me, but it has helped me to emerge from my grief with a sense of purpose and agency. Since 2011, I've felt invested in their impactful work to stop Alzheimer's.

"Inspired by others who raised money for CureAlz year after year, I always intended to step up again. In 2024 I had that opportunity: a 50-state ultra-trail running adventure. Runners joined me in several states, themselves motivated to end Alzheimer's. Most glorious of all was running with my adult daughters, not something possible for my dad, and easily among my life's greatest joys.

"I feel like we are on the cusp of breakthroughs and, this year, CureAlz's work identifying and supporting critical projects feels more critical than ever. Quite simply: CureAlz gives me hope. Thank you!"



DAVID WILKINS

"My wife of 56 years, LaNell, was exhibiting signs of cognitive decline for years before she was diagnosed with Alzheimer's," says Dave Wilkins. "At first, I did not recognize the symptoms. Eventually, I set up appointments with neurologists, though it was always difficult to get her to go. Over time, our children encouraged us to move closer to them, and finally, we made the move in the fall of 2022.



"Our move triggered the start of a difficult period for LaNell. After multiple trips to emergency rooms, hospital visits and a brief stay in a memory care facility, I ultimately brought her home in November. Having LaNell at home was both very rewarding and comforting for me. She passed one year later, in November of 2023.

"LaNell's mother, Orlena, was also affected by Alzheimer's disease. She passed shortly after her daughter in December of 2023. My in-laws, despite having minimal formal education, were very smart and frugal. They always saved well and lived below their means. When they passed, LaNell's siblings made sure I received a sizeable share of the estate, which I directed to Cure Alzheimer's Fund.

"My nephew, who has a Ph.D. in neuroscience, helped me discover the organization. Together, as a family, we decided research would be the best way to honor LaNell and Orlena's memories.

"If Alzheimer's disease is to be conquered, it will be through research."

JERRY PORZEMSKY "My father suffered with Alzheimer's and that motivated me, a practicing psychologist, to do all I could to prevent this fate from [happening to] myself and others," says Jerry Porzemsky. "I have maintained an active lifestyle, adventurously traveling doing tribal photography, spending time with friends, vigorously exercising daily and keeping myself engaged with the joys of lifemusic, art, nature, etc. I began donating to the Cure Alzheimer's Fund 13 years ago and have included this wonderful charity in my estate plans. I truly believe that research is the only solution toward human freedom from Alzheimer's, and especially at this time when governmental support is questionable."

Jerry Porzemsky (right)

HAMMY REL13F FUND

"I always looked up to my great-grandfather. He was energetic and so full of life," says Jimmy Hamiton. "He inspired our connection to Cure Alzheimer's Fund. I wanted to raise awareness while encouraging more young adults to engage in charity work and the fight against Alzheimer's.



From left: Nick Forbes, Jake Collett, Jimmy Hamilton, Brianna Hamilton, Christopher Hamilton, Dom Ciardullo and Aidan Frank.

"Each year, Hammy Rel13f Fund hosts a golf outing to raise funds for Alzheimer's research. We are incredibly proud of our board, all of whom are 30 years old or younger, for putting on this effort. Choosing CureAlz as our partner was an easy decision—100% of the funds we raise go directly to research, which is crucial to finding a cure.

"At our golf outing this past year, we were honored to recognize the Melillo family, who lost their beloved Nicola Melillo to Alzheimer's. Their story reminds us why we do this important work.

"Thanks to the continued support of our donors, golfers and sponsors," we've raised over \$109,000 in just four years. We are excited to continue our work and look forward to making an even greater impact in the years to come."

ASHTON AND DAVID HARRISON

"To watch someone you love go from being the smartest, most wonderful person you've ever known to someone who can't walk, talk or function is a painful journey," says Ashton Harrison. "Although it can be a lonely, overwhelming and exhausting experience, and you don't have your loved one to confide in and lean on to help with such an overbearing task, you will survive this very difficult time. The good news is, they will never stop knowing that you are 'their person.'

"I have been an avid supporter of Cure Alzheimer's Fund since it is unbiased in its approach to the science and all funds go to research," she says. "Their panel of consultants is the best in the industry and always on the cutting edge. The management team is so impressive and most have a personal connection to the disease. I believe this is the only way we will protect our kids and future generations from going through this journey."



Ashton lost her husband of 36 years to Alzheimer's in April 2022.



TARA MANUFACTURING INC.

In 2024, Tara Manufacturing, a leading innovator in pool and outdoor products, dedicated all profits from the sale of its Celestial Skies pool liner pattern to Alzheimer's research. The gift was made in honor of the company's founder, Marshall Richardson, as well as employees and loved ones who have been personally impacted by the disease.

In September, Tara Manufacturing presented Cure Alzheimer's Fund with the largest philanthropic gift in the company's history: \$338,384.01.

"We are incredibly proud of the Celestial Skies initiative and what it has accomplished in support of Alzheimer's research," said Tara Furlough, CEO of Tara Manufacturing. "The dedication of our employees, partners and customers has made this donation possible, and we're honored to play a part in helping find a cure." The Celestial Skies initiative was made possible through a partnership between Tara Manufacturing and i2M, a leading manufacturer of specialized materials. CEO Alex Grover and her team at i2M donated an additional \$5,000 to Cure Alzheimer's Fund.

The team at Tara Manufacturing hopes that the Celestial Skies initiative will inspire others to stand with them and make a difference. Their donation to Cure Alzheimer's Fund is a testament to the company's commitment to social responsibility, and its belief in the power of innovation to make a lasting impact.

Shining a Light on Alzheimer's Disease: Amplifying Our Mission

Cure Alzheimer's Fund remains committed to advancing the understanding of Alzheimer's disease (AD) by sharing the progress made possible through our research grants. In 2024, we expanded our awareness campaign across new media platforms, hosted events and provided a forum for our researchers to present the latest advancements in our scientific understanding of AD.

Webinars

The CureAlz webinar series serves as a vital resource for those seeking to understand the complexities of Alzheimer's disease research. These presentations deliver comprehensive updates while offering valuable insights into cutting-edge scientific advancements. Throughout 2024, we presented seven webinars exploring critical aspects of Alzheimer's, including brain aging, inflammation, the neuroimmune response, sleep and circadian rhythm, and the puzzle of sex and gender, among other compelling topics. We invite you to access our complete library of presentations for on-demand viewing at **CureAlz.org/news-and-events/webinars/**.















Webinar presenters, L to R: Murali Doraiswamy, MBBS, FRCP; Julie Harris, Ph.D.; Shane A. Liddelow, Ph.D.; Erik S. Musiek, M.D., Ph.D.; Miranda Orr, Ph.D.; Beth Stevens, Ph.D.; and Rudolph E. Tanzi, Ph.D.

Founders Dinner

A dinner celebrating our Founders on the 20th anniversary of Cure Alzheimer's Fund was hosted at Raffles Boston. During this meaningful occasion, Rudolph E. Tanzi, Ph.D., presented an insightful 20-year retrospective on the evolution of Alzheimer's research, reflecting on significant milestones and the path forward.



From left: Rudolph E. Tanzi, Ph.D, Jacqueline Morby, Tim Armour, Phyllis Rappaport and Henry McCance.

Public Awareness

In 2024, Cure Alzheimer's
Fund amplified its message
on the critical need for
Alzheimer's research, reaching
broader audiences through
dynamic new channels. From
captivating billboards to
compelling radio segments



and our newly launched publication, "CEO Insights and Updates," we expanded awareness to more people than ever before.



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.

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

CureAlz Legacy Society

Join the Legacy Society by including Cure Alzheimer's Fund in your will or estate plans, ensuring your generosity endures beyond your lifetime. In 2024, an incredible 29% of our research support was made possible by the Legacy Society.

To learn more, email us at Legacy@CureAlz.org.

Tax-Smart Ways to Give

There are many tax-efficient ways to make a lasting impact—now and beyond your lifetime.

Qualified Charitable Distribution (QCD)

If you're 70½ or older, a QCD paid from your Individual Retirement Account (IRA) allows you to donate up to \$108,000 to Cure Alzheimer's Fund. And it's 100% tax free.

Retirement Assets or Life Insurance Policies

You can make a gift of retirement assets such as an IRA, a 401(k) and life insurance proceeds by naming Cure Alzheimer's Fund as a beneficiary.

Charitable Gift Annuities and Trusts

Charitable gift annuities and trusts can provide current or deferred income to a donor or beneficiary and, ultimately, a contribution to Cure Alzheimer's Fund.

Donor Advised Funds (DAF)

Recommend a donation to Cure Alzheimer's Fund through your donor advised fund. Contact us to learn more about the benefits of a DAF.

To learn more about these and other tax-efficient ways to maximize your generosity, email us at Info@CureAlz.org.





Make a One-Time or Recurring Gift Online

Make a secure gift online by credit card, PayPal, Venmo or cryptocurrency. Visit **Give.CureAlz.org/2024annualreport.**



Securities and Wire Transfer

Securities such as stocks, mutual fund shares or other appreciated assets can be a beneficial way to support Cure Alzheimer's Fund while potentially reducing your tax burden. Gifts may be sent electronically via wire transfer. Please contact Laurel Lyle at LLyle@CureAlz.org.



Mail

Make your check payable to Cure Alzheimer's Fund.



Telephone

To donate by telephone, call **781-237-3800**. Our business hours are 9 a.m. – 5 p.m. ET.



Start a Community Fundraiser

From bake sales, road races and livestreams to everything in between, you can turn any passion into a fundraising activity. Email **Hero@CureAlz.org** to share your idea with us and receive a copy of our fundraising guide.

DONATE TODAY



To explore these and other ways to give, scan the QR code, visit **Give.CureAlz.org/2024annualreport**, email us at **Info@CureAlz.org** or call **781-237-3800**.

RECOGNIZED FOR EXCELLENCE



Cure Alzheimer's Fund has achieved a 100% perfect score and the highest rating of 4 stars for 13 consecutive years.



Cure Alzheimer's Fund has received the designation of Platinum level, the highest recognition offered by GuideStar.



Cure Alzheimer's Fund meets all 20 Better Business Bureau Standards for Charity Accountability.





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CureAlz.org



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