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 2023

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Dear Friends,

Thanks to all of you, 2023 was another record-breaking year for Cure Alzheimer’s Fund.

We funded more projects (114) and investigators (147) than ever before and achieved a new high total for our research program of just more than $27.8 million. We also set ourselves up to surpass $200 million in research spending since inception in 2024. What a wonderful way to enter our 20th year of funding research to solve the mysteries of Alzheimer’s science and accelerate progress toward treatments and a cure.

I am so grateful for the successes of 2023, particularly because it was also a year of change for the organization.

A YEAR OF CHANGE

On Sept. 2, 2023, Jeffrey L. Morby, who founded our organization in 2004 along with his wife and CureAlz Board member Jacqui, passed away. Jeff was a visionary, a successful businessman, a philanthropist, a loving husband and father, and a dear friend. He was passionate about eliminating Alzheimer’s disease and improving the lives of millions of people, always deeply engaging with the science we fund and challenging our scientists to answer the toughest questions. Jeff was a founding Board member, serving as Chairman since inception and my Co-Chair since May 2016. His presence and guidance are truly missed. We mourn Jeff’s loss and are determined to further his legacy of pursuing the vital answers to the mysteries of Alzheimer’s disease.

To honor Jeff’s memory, the Board of Directors of Cure Alzheimer’s Fund has established the Jeffrey L. Morby Prize. This annual prize, which has not yet been awarded as our annual report goes to press, recognizes the key authors of a recent CureAlz-supported research publication that, in the opinion of leading scientists, transforms our fundamental understanding of Alzheimer’s disease and opens new paths to translate scientific results into effective ways to prevent, diagnose or treat Alzheimer’s disease.

This year, Tim Armour, our founding President and CEO, retired after 18 years with Cure Alzheimer’s Fund. In 2005, Tim accepted the challenge to lead our start-up and our mission as our first CEO. His exceptional commitment and stewardship grew Cure Alzheimer’s Fund into the thriving organization it is today. We are grateful for all that he has done, and that he continues to hold a seat on the Board of Directors.

The Board prioritized selecting a successor to Tim with extensive knowledge of the science of Alzheimer’s. After a thorough search process, utilizing the services of a national executive search firm, it became absolutely clear that the best person to guide our future was already among us. Meg Smith, who has led our research funding program since 2015, became our new CEO on Oct. 1. During Meg’s tenure managing the research program, CureAlz awarded $138 million in research grants—nearly 80% of total distributions since our founding in 2004. She spearheaded this growth while simultaneously ensuring that CureAlz maintained its model of high-rigor, high-accountability scientific decision making by its external community of leading researchers. Previously
trained as an attorney and with experience in business consulting, Meg earned tremendous respect within the research community in her role, particularly impressive for a nonscientist. The Board of Directors has complete confidence that Meg’s experience, professionalism, spirit of collaboration and strategic insights will be a catalyst for our continued growth.

This is an incredible amount of change for any organization, and I am proud to say that our small but mighty team remained steadfast in their work and focused on our mission. The transition was smooth, and we finished 2023 with an 8% increase in total fundraising and a 13.6% increase in research outflows from the prior year.

PROGRESS TOWARD A CURE
On July 6, 2023, the U.S. Food and Drug Administration (FDA) granted traditional (full) approval for the drug Leqembi® (the brand name for lecanemab) for Alzheimer's disease. While Leqembi is only of benefit to those in the early stages of the disease, it should be celebrated as real progress—and the beginning of future treatments for all stages of Alzheimer’s disease.

Since our inception through the end of 2023, 1,177 papers have been authored thanks to CureAlz funding, an indication of just how groundbreaking the extraordinary work we have supported over the last 19 years is. These papers have been cited in turn more than 97,000 times by other scientists in their own publications—validation of our model and intent for our funding to accelerate progress across the entire scientific community.

In 2023, we realized a 7.8x return on our investment into proof-of-concept research from the combined years 2018 to 2021, the latest data available. The total of $64 million in grants provided to researchers from Cure Alzheimer’s Fund in those years resulted in an extraordinary $497 million of follow-on funding from the National Institutes of Health (NIH)/National Institute on Aging (NIA). It is wonderful to see our support of bold bets and novel hypotheses flourish.

IN GRATITUDE
Progress toward finding a cure would not happen without so many believing that a cure is possible. Our shared belief gets us halfway there; our shared action will get us across the finish line.

My deepest gratitude to the many partners of Cure Alzheimer’s Fund during the last 19 years: our incredible researchers who inspire us with their creativity, dedication and spirit of inquiry; our Board of Directors, Trustees and key donors who fund operations so 100% of general donations can go to support our research program; and the amazing CureAlz team that masterfully runs the operation moving us closer to our goal.

And, of course, I am truly grateful for all of you, the more than 73,000 generous donors who have chosen to support research through Cure Alzheimer’s Fund over the last 19 years. I know how personal this fight is for many of you. It is also very personal for me. In January 2023, I lost my wife, Allison, after a 22-year battle with Alzheimer’s. Together, our shared experience and collective determination will propel us across the finish line.

Sincerely,

Henry F. McCance
Chair, Board of Directors
Honoring Jeffrey L. Morby
1937 – 2023

Creating a true, lasting difference in the world is an immense achievement.

Since our founding in 2004, the researchers, donors, Board members, Trustees and staff of Cure Alzheimer’s Fund have had the opportunity to contribute their time and effort to make such a difference due directly to the vision and devotion of our Co-Founder and Co-Chair Jeffrey L. Morby. Along with his wife, Jacqui, Jeff started our organization and provided its initial inspiration of eliminating Alzheimer’s disease to relieve the physical, emotional and financial burden of the illness for millions of people throughout the world.
In addition to his crucial role in founding Cure Alzheimer’s Fund, Jeff was a successful business leader, philanthropist, loving husband, caring father and dear friend. We offer our deepest sympathies to his family, especially his lifelong love and wife, Jacqui, who lost her life partner of 64 years.

We also share our gratitude.

The relentless passion Jeff had for understanding Alzheimer’s disease motivated our organization to play a key role in some of the most important scientific discoveries about the brain and the disease. He asked insightful and probing questions about the science. He had tremendous respect for the researchers who commit their lives to the cause. And he developed a profound knowledge of the science, contributing suggestions for novel ways to investigate the disease.

As described by Dr. Rudy Tanzi, Chair of the Cure Alzheimer’s Fund Research Leadership Group,

“Jeff was a pioneer, visionary and tremendous catalyst for the progress Cure Alzheimer’s Fund has made in driving so many seminal paradigm shifts in our understanding of Alzheimer’s disease. From our first meeting almost 20 years ago, he was passionate about science and knew research was the only way to end the burden of this disease for patients and families. Jeff was driven to ensure that the highest-impact research would receive funding with the best Alzheimer’s researchers in the world brought together to collaborate and share their discoveries. His commitment to finding a cure continues to inspire the groundbreaking work of the many researchers supported by CureAlz throughout the world.”

We mourn this significant loss—and remain steadfast in our efforts so that we may respect Jeff’s legacy. We continue our work to advance the science of Alzheimer’s disease, and will persist until his goal of finding a cure is realized. It is our honor to continue the work that Jeff inspired and guided.
Our People

BOARD OF DIRECTORS

HENRY F. McCANCE
Chair, Board of Directors, and Founding Board Member
Chairman Emeritus, Greylock Partners
Partner, Fenway Sports Group
Trustee, McCance Family Foundation

ROBERT F. GREENHILL
Founder, Greenhill & Company

JACQUELINE C. MORBY
Founding Board Member
Senior Advisor, TA Associates
Chair, Morby Family Charitable Foundation

TIM ARMOUR
Former President and Chief Executive Officer, Cure Alzheimer’s Fund

PHYLLIS RAPPAPORT
Treasurer
Founding Board Member
Chair, Phyllis and Jerome Lyle Rappaport Charitable Foundation
Director, New Boston Fund Inc.

RICHARD BIRNBAUM
Chairman and Director, Rick Sharp Alzheimer’s Foundation

MEG SMITH
Chief Executive Officer, Cure Alzheimer’s Fund

ADMINISTRATION

JO ANTONELLI
Chief Financial Officer

TAMMY AWTY, PH.D.
Science Communicator

LISA BIDA
Marketing Manager

BARBARA CHAMBERS
Executive Vice President, Marketing and Communications

KYRSTEN CONOVER
Development Associate, Operations

INGRID DANKERS
Gift Processing Assistant

DANNY HARPER
Senior Philanthropic Advisor

JULIE HARRIS, PH.D.
Executive Vice President, Research Management

MAHUA HEATH
Senior Philanthropic Advisor

HEIDI HENDERSON
Controller

MORGAN HERMAN
Executive Vice President, Development

LAURA HILL
Development Operations Assistant

LAUREL LYLE
Vice President, Board Relations and Development Operations

LORI MARCHETTI
Accounting Supervisor

LAINIE MORRIS
Senior Development Associate

CHRISTINA NOVAK
Senior Philanthropic Advisor, Institutional Relations

LORI PRESCOTT
Accounting Assistant

LISA RAND
Vice President, Marketing and Communications

EMANUELA ZAHARIEVA RAPPAPORT, PH.D.
Science Communicator

CAITLIN SAIA
Director, Grant Administration

CHARLES SEAKS, PH.D.
Director, Grant Management

NIKKI SENGSAVANH
Director, Development Operations

SHARI CROTTY
Trustee, The Crotty Family Foundation

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

TRUSTEES

KATHLEEN ARNOLD
Trustee, Fleming Foundation

JONO BACON
Founder, Community Leadership Core

ANOOSHEH BOSTANI
Director, Alfred E. Mann Charities

JEFF AND BLAKELEY BURATTO

MARK FAGGIANO
Founder and Former CEO of TaxJar, acquired by Stripe

SHARI CROTTY
Trustee, The Crotty Family Foundation

CHRISTINA KOhnen
Trustee, Kohnen Family Foundation

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

KUMAR MAHADEVA
Founder and Former CEO, Cognizant Technology Solutions

JEROME MAZURSKY
Founder, Mazursky Group

EDIE AND JOHN RANDALL

CHRISTINE VILLAS-BOAS
President, Michel & Claire Gudefin Family Foundation
Tim Armour joined Cure Alzheimer’s Fund as the first President and Chief Executive Officer in 2005 shortly after our founding, and was at the helm and heart of our organization for 18 years until his retirement in October 2023.

Tim was the first employee to join CureAlz. In his role, Tim managed the growth of our organization and built a small but mighty team. One of his gifts is identifying the right people with the right expertise to serve our important mission. Tim fostered relationships with each staff member with his earnest humor, unwavering conviction and genuine optimism that, through research, there will be a cure for Alzheimer’s disease.

During Tim’s tenure as CEO, CureAlz distributed more than $175 million to research into understanding the causes leading to Alzheimer’s disease. The progress resulting from this funding is considerable, and a testimony to the purpose and responsibility he felt to lead the organization and move us closer to a cure.

The connections Tim made with donors were genuine and deeply personal. He valued understanding their experiences with Alzheimer’s disease, whether it was memorializing their loved ones or contributing to research with the hope of sparing the next generation from the devastation caused by the disease.

“On behalf of our Board of Directors, I want to thank Tim for his leadership of Cure Alzheimer’s Fund and his dedication to our mission,” said Henry McCance, Board Chair. “Because of Tim’s commitment, we are making tremendous progress with understanding this disease.”

We are immensely grateful for all that Tim has contributed to Cure Alzheimer’s Fund during his tenure as President and CEO, and look forward to his continued contributions as a member of our Board.
6.9 million Americans have Alzheimer’s disease, and that number is expected to double by 2050. Loved ones and communities lose people every day they need and value. What an honor it is to be chosen to lead an organization with the incredible mission of finding a cure.

During the last nine years, I have had the privilege of working directly with scientists from around the world who are dedicated to unraveling the causes of Alzheimer’s and developing effective therapies and methods of prevention. To be able to report that in 2023 CureAlz funded 147 researchers in 11 countries with a new record for total research spending of $27.8 million both gratifies and humbles me. The project delays and extensions of COVID-19 are behind us and incredible progress is being made in our fully operational research labs around the world.

2023 was an extraordinary year marked by the first regular FDA approval of a disease-modifying therapy for mild cognitive impairment due to Alzheimer’s disease. Leqembi®, developed by pharmaceutical companies Eisai and Biogen, is designed to reduce the amount of aggregated amyloid beta in the brain. In Leqembi’s Phase 3 clinical trial, treated participants experienced cognitive decline at a 27% slower rate than that experienced by control participants. Thus far, this class of drug is of benefit only to those in early stages of Alzheimer’s disease, and it achieves a slowing of the progression of the disease and not a reversal. Treatment is administered intravenously and carries the risk of serious side effects. Despite these limitations, Leqembi should be celebrated for the real progress it represents as we continue to work for improved therapies in the future.

Leqembi and the other drugs in its class essentially emulate naturally occurring proteins. Our bodies generate them in response to the presence of amyloid beta in order to trigger clearance by the brain’s innate immune system. One of the key insights that led to these drugs can be traced back to a 2005 paper from CureAlz-affiliated scientists Drs. Rudy Tanzi and Rob Moir. However, numerous previous Phase 3 clinical trials of similar drugs removed amyloid beta but failed to achieve clinical benefits. The story of Leqembi, and other drugs like it, reminds us that medical progress can be very slow and in anything but a straight line—but every failure we have experienced as a field has helped us get closer to this day, and to the future day when we have effective therapies for everyone at every stage of Alzheimer’s disease.

The Amyloid Cascade Hypothesis of Alzheimer’s Disease postulates that aggregation and accumulation of misfolded amyloid beta protein in the brain triggers downstream consequences that include tau pathology, neuroinflammation, neurodegeneration and cognitive decline. The failure of past clinical trials of anti-amyloid therapeutics challenged this hypothesis and led many industry participants to abandon amyloid as a target. However, data from across the CureAlz portfolio of scientific projects has consistently supported the centrality of amyloid, and so CureAlz has continued to fund projects focused on amyloid and its role within the cascade.
The benefit to patients seen in trials of recent anti-amyloid drugs like Leqembi is gratifying reinforcement of our founding principle to always follow the science. While the challenges of this complex disease are many, CureAlz and our scientific community are energized by our progress and ready to continue to push forward.

Achievements like this have long been imagined by our organization’s Founders. Sadly, this year we lost our Co-Chair and Founder Jeff Morby, whose vision, determination and drive contributed greatly toward the progress we now are experiencing. I miss his incisive questions and fierce insistence that CureAlz always find and fund the best science and the best scientists. While we mourn this significant loss, we continue the work that he inspired with enthusiasm and conviction.

As CEO, I get to experience Cure Alzheimer’s Fund in a new way. I work more closely with the entire CureAlz team, who have always impressed me with their expertise, diligence and dedication to our mission. In 2023, we promoted our Controller, Jo Antonellis, to Chief Financial Officer, and Julie Harris, Ph.D., to the role of Executive Vice President, Research Management. Our research team is growing in numbers and scientific training and, under Julie’s leadership, they are maintaining our founding principle that all of our scientific decisions are made by leading scientists active in the field.

We have also been able to maintain our founding principle that all general donations go to our research program thanks to operational support from our Board, Trustees and certain other donors who support Cure Alzheimer’s Fund’s operations. I am grateful to them for enabling our team to share our message, raise funds and act as responsible stewards for general donations. I value the opportunity to be accountable to their high standards and celebrate that their $5.8 million in Foundation Fundraising in 2023 translated into $29 million for General Fundraising, a 5x multiplier that will drive our 2024 research program.

One of the most extraordinary benefits of my new role is that I get to meet more of you, our partners, who in 2023 donated a record-breaking $34.8 million through more than 22,000 gifts. Hearing about the deep impact Alzheimer’s has had on you and your families is meaningful and inspirational to me and the entire CureAlz team. In 2024, we will bring more science and scientists to you through online and other events and on our website; I hope you will meet us there.

Thank you for joining Cure Alzheimer’s Fund in the fight against Alzheimer’s disease. We celebrate the progress that has been made while staying determined to enable the research necessary to answer the vital questions that remain.

Warmly,

Meg Smith
CEO

“To be able to report that in 2023 CureAlz funded 147 researchers in 11 countries with a new record for total research spending of $27.8 million both gratifies and humbles me.”
Alzheimer’s disease has no cure. Yet.

6.9 MILLION
Americans living with Alzheimer’s disease

$360 BILLION
Projected cost of Alzheimer’s and other dementias to the United States in 2024

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

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 disease-modifying 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.

Source: Alzheimer’s Association, 2024 Alzheimer’s Disease Facts and Figures
Enabling the world’s leading scientists to explore bold ideas and make game-changing discoveries.

We can do this because of you.

73,000+ DONORS
$193,000,000+ GENERAL DONATIONS TO SUPPORT OUR RESEARCH PROGRAM

Together we are making an impact.

807 RESEARCH GRANTS
$193M RESEARCH FUNDS DISTRIBUTED
1,177 RESEARCH PAPERS PUBLISHED
97,674 RESEARCH PAPER CITATIONS

7.8x CureAlz’s research investment of $64M from 2018–2021 yielded $497M in funding from 2018–2023 from the National Institutes of Health—a 7.8x return on investment.

2023 Source and Use of Funds

In 2023, Cure Alzheimer’s Fund received $5,790,729 in Foundation Fundraising (FFR) from our Board of Directors, Trustees and a core group of other donors to support our operations. $29,004,854 in General Fundraising (GFR) was received to be applied to support our research programs. Our Total Fundraising (TFR) for 2023 was $34,795,583.

**Source of Funds**

- **$34,795,583**
  - Individuals: $14,544,060 (41.8%)
  - Founders/Board of Directors/Trustees/Core Group of Other Donors for FFR: $5,790,729 (16.7%)
  - Foundations: $3,762,570 (10.8%)
  - Corporations: $358,091 (1%)
  - Trusts/Estates: $10,340,133 (29.7%)

**Use of Funds**

- **$34,618,691**
  - Research Distributions and Support: $27,793,083 (80.3%)
  - Fundraising: $1,897,310 (5.5%)
  - Management and General: $1,320,840 (3.8%)
  - Programs: $3,607,458 (10.4%)

Source and Use of Funds obtained from internal records.
### Statement of Financial Position

**Assets**

**Current Assets:**
- Cash and cash equivalents: $5,212,810
- Pledges receivable, current portion: 2,450,253
- Investments: 24,112,740
- Prepaid expenses and other current assets: 239,429
  - **Total current assets:** $32,015,232

- Pledges receivable, less current portion, net: 1,332,899
- Right-of-Use, Asset, net: 140,148
  - **Total Assets:** $33,488,279

**Liabilities and Net Assets**

**Current Liabilities:**
- Current portion of operating leases payable: $125,706
- Accounts payable: 316,299
- Research grants payable: 718,427
- Accrued payroll and related: 566,892
  - **Total liabilities:** $1,727,324

**Net Assets:**
- Without donor restrictions: 27,791,118
- With donor restrictions: 3,969,837
  - **Total net assets:** $31,760,955

**Total Liabilities and Net Assets:** $33,488,279

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### Statement of Cash Flows

**Cash Flows from Operating Activities:**

**Cash received from:**
- Contributions: $26,479,555
- Investment income: 977,403
  - **Total receipts:** $27,456,958

**Cash paid for:**
- Research distributions and support: (27,562,916)
- Salaries and related expenses: (4,427,507)
- Professional fees: (1,185,498)
- Gift processing fees: (146,420)
- Occupancy expenses: (213,989)
  - **Other expenses:** (1,005,040)
  - **Total expenditures:** (34,541,370)
  - **Net cash provided (used) by operating activities:** (7,084,412)

**Cash Flows from Investing Activities:**
- Proceeds from sale of investments: 8,426,908
- Purchase of investments: (6,173,178)
  - **Net cash provided (used) by investing activities:** 2,263,730

**Net Increase (Decrease) in Cash and Cash Equivalents:** (4,820,682)
- **Cash and Cash Equivalents, beginning of year:** 10,033,492
- **Cash and Cash Equivalents, end of year:** 5,212,810

**Noncash Operating and Investing Activity:**
- Donated stock: $8,382,433

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**Statement of Activities**

**Revenue and Support:**
- Contributions: $28,115,172
- Donated stock: 8,382,433
- Special events, net of direct expenses: 185,317
- Investment income, net: 1,238,114
  - **Total revenue and support:** 37,921,036

**Expenses:**
- **Program:**
  - Research distributions and support: 27,793,083
  - Other program expenses: 3,607,458
  - **Total program expenses:** 31,400,541
- Management and general: 1,320,840
- Fundraising: 1,897,310
  - **Total expenses:** 34,618,691

**Change in net assets:** 3,302,345
- **Net Assets, beginning of year:** 28,458,610
- **Net Assets, end of year:** $31,760,955

Source: Audited Financial Statements
Cure Alzheimer’s Fund’s investment in research continues to be driven by strong increases in overall contributions.
As I look back on 2023, it was a truly remarkable year.

As the Chair of the Research Leadership Group, I have the privilege of working with the most extraordinary group of researchers who have dedicated their professional lives to taking on the demands of solving what is arguably the most challenging disease facing humanity: Alzheimer’s disease. Deciphering and intervening in the slow destruction of the brain and loss of self requires a rigorous scientific process with a community of researchers who embody fortitude, determination, patience—and even fearlessness.

Throughout 2023, the milestone statistics that unfolded as a result of the research grants provided by CureAlz were gratifying for all of us. The number of published papers in key, significant science journals and the citations in 2023 and cumulatively are evidence of how CureAlz-funded research is deepening the entire field’s understanding of the underlying causes and triggers of the pathology; this already is leading to therapeutic solutions for Alzheimer’s. While the statistics are meaningful, the discoveries made with the funding provided by CureAlz are even more remarkable.

The approval of Leqembi®, a drug targeting amyloid in patients with early-stage Alzheimer’s, is just the first of many therapies that will be developed for patients at all stages of the disease, including prevention. The approval of this drug is evidence of the importance of amyloid as the key driver of the pathology cascade of Alzheimer’s disease.

While I reflect on the year and the continued outstanding accomplishments of this organization and the scientists it supports, I also am reminded that much of this only happened thanks to the vision and perseverance of our Co-Founder, Jeff Morby. Jeff had a lifelong fascination with the human brain and a passion for finding a cure for Alzheimer’s disease. He had great respect for the researchers in the field and was a dedicated advocate for those who have Alzheimer’s. I was honored to call him a friend.

Many of us have lost someone to this disease. In 2023, we also lost Allison McCance, who battled Alzheimer’s disease with dignity and grace for 20 years. In Allison, we were reminded of the desperate need to find solutions for those who are battling with the disease; in her husband—our Co-Founder—Henry’s example, we see how love and commitment can through grace transform into a benefit to many others facing the same challenging circumstances. In honor of Jeff and Allison, we continue the work started nearly 20 years ago—the vitally important and meaningful mission of finding a cure for Alzheimer’s disease.

Rudolph E. Tanzi, Ph.D.
Chair, Research Leadership Group
Cure Alzheimer’s Fund set a record for scientific funding in 2023. We deployed $27.8 million for research purposes, a 13.6% increase from the previous record set in 2022 ($24.5 million). This total represents funds in support of 114 grants and 147 named investigators.

A fundamental principle of CureAlz’s approach to accelerating science to benefit the Alzheimer’s disease community is to provide funding where other entities will not, and prioritize returns measured in scientific progress, not potential profits. Thus, we continue to prioritize basic Alzheimer’s disease research over drug discovery, with most distributions in 2023—94% of awards—directed to early-stage discovery research.

Most funding went to translational research projects and a large fraction, around 25%, of all funded studies are on the immune response 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 represented less than 5% of funding in 2023. We funded a healthy balance of renewing projects and entirely new or follow-on projects as part of our portfolio.

“The support from Cure Alzheimer’s Fund has enabled us to explore high-risk, high-reward topics that would not be funded by more traditional funding mechanisms, but hold the potential to make a transformative difference in both our understanding as well as the treatment of Alzheimer’s disease.”

—JOHN R. LUKENS, PH.D.
More important than these numbers are the evident progress and wonderful discoveries enabled by this funding. 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.
**Consortia Model**

Cure Alzheimer’s Fund historically has supported innovative research projects, with grants at the $100,000 to $300,000 level, that have the potential to add significant new understanding of Alzheimer’s pathology to the field. While CureAlz continues to enthusiastically support these standalone high-risk, high-potential efforts, advances in understanding Alzheimer’s disease have warranted larger-scale investigations within specific areas of the science. The consortia model provides for organized, large-scale initiatives with an expanded level of collaboration and shared cross-institutional goals.

In 2023, 24% of research spending went toward five active consortia. Each consortium had a regular schedule of meetings throughout the year that brought lab leaders and personnel together to discuss progress, share findings, offer encouragement and ensure productive collaborations. After several grant cycles, this year we closed the Alzheimer’s Disease Drug Discovery and Development (AD4) and the Collaboration to Infer Regulatory Circuits and Uncover Innovative Therapeutic Strategies (CIRCUITS) consortia and transitioned some of the projects to standalone awards. We launched a “refreshed” Neuroimmune Consortium and a new Microbiome Consortium.

**ALZHEIMER’S DISEASE TAU CONSORTIUM**

A considerable amount is known about the involvement of tau in neurodegenerative diseases where it is the main contributing pathology, known as primary tauopathies. However, tau’s role in Alzheimer’s disease, a secondary tauopathy in which tau pathology develops in an AD-specific pattern in response to amyloid beta, is less clear. Given that tau is so closely associated with neurodegeneration and cognitive impairment in AD, a deeper understanding is necessary to enable improved diagnostic and prognostic biomarkers, as well as to identify new opportunities for effective therapeutic intervention. The Alzheimer’s Disease Tau Consortium brings together leaders with the expertise and motivation to shed light on tau’s complex role specifically in Alzheimer’s disease.

**FLEMING APOE (APOLIPOPROTEIN E) CONSORTIUM**

Substantial data inform us that APOE gene variants differentially affect risk for developing sporadic Alzheimer’s disease, yet the mechanisms that explain this are not understood. Increased risk may arise from how APOE affects amyloid plaque accumulation and clearance, tau tangle formation, neuroinflammation, non-Alzheimer’s disease hallmarks of aging and the porosity of the blood-brain barrier. The Fleming APOE Consortium brings together experts to develop a better understanding of these mechanisms.
BRAIN ENTRY AND EXIT CONSORTIUM
The brain is a remarkable but fragile organ with limited ability for self-renewal following injury; it also has a very high metabolism, using approximately 20% of all energy consumed by the body and producing significant waste. Consequently, it has evolved a complex system of barriers to control the entry and exit of materials and maintain its delicate healthy balance. The Brain Entry and Exit Consortium is investigating how each component of the brain’s entry and exit structures, along with the cerebrospinal fluid that flows through the brain, must function together to maintain health.

MICROBIOME CONSORTIUM
The gut microbiome is the community of trillions of microorganisms, including bacteria, fungi and viruses, that live in the digestive tract. These microbes play an essential role in maintaining our health, and emerging research suggests there may be a link between the gut microbiome and Alzheimer’s disease. The Microbiome Consortium represents a group of world-renowned Alzheimer’s disease (AD) researchers who will take a multifaceted approach to investigating the mechanisms by which the microbiome impacts Alzheimer’s disease. Work by the consortium also includes uncovering the full spectrum of microbes that may be present in the AD brain to understand the potential role of infections driving AD pathology.

NEUROIMMUNE CONSORTIUM
Neuroinflammation is a well-known feature of Alzheimer’s disease dating back to the description of reactive glial cells in the brain by Alois Alzheimer more than 100 years ago. Modern research shows that this inflammatory response is not just an innocent bystander during disease progression. Many genetic risk factors for sporadic Alzheimer’s disease affect the brain’s immune cells—microglia and astrocytes. The researchers in the Neuroimmune Consortium are mapping out the roles of these and other immune cells in the brain during Alzheimer’s disease.

“Cure Alzheimer’s Fund has enabled us to take big risks on new ideas, speeding movement into new and novel spaces for research and accelerating science out of the lab and (ultimately) into the clinic. Even more important is the expansive network of Alzheimer’s disease experts that are always on hand to provide insights, advice and support to push these discoveries ever further forward.”

—SHANE A. LIDDELOW, PH.D.
“Cure Alzheimer’s Fund support sparked a collaboration between two investigators who on the surface appear to study two totally different topics. Together we teamed up to think about Alzheimer’s disease from a new perspective.”
—ANNA GREKA, M.D., PH.D.

Advisory Groups and Meetings
The collaborative efforts at Cure Alzheimer’s Fund also include groups of researchers working together to help set priorities and drive the direction of research distributions. Two advisory groups, made up of esteemed researchers, share their expertise and participate in several meetings throughout the year to facilitate collaboration and disseminate research findings to the broader community.

■ RESEARCH LEADERSHIP GROUP (RLG)
The 2023 RLG included 39 leading scientists specializing in Alzheimer’s disease and related fields. These leaders are the primary decision makers regarding our overall direction, as well as whether to support specific proposals and projects. The RLG refers investigators, conducts peer reviews on research proposals and reports, participates in quarterly meetings and drives collaboration.

■ SCIENTIFIC ADVISORY BOARD (SAB)
The role of the SAB is to provide guidance to Cure Alzheimer’s Fund regarding our overall scientific direction and funding impact. 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 benefits for patients.

■ INTERNATIONAL CONFERENCE ATTENDANCE AND SPONSORSHIP
Cure Alzheimer’s Fund was a sponsor of the International Conference on Alzheimer’s Disease and Parkinson’s Diseases (AD/PD) held in March 2023 in Gothenburg, Sweden. This annual conference is the largest global conference dedicated to basic science and translational and clinical research, and featured many of our funded researchers and projects.
Published Papers

Once a researcher has completed their work, they document the details and outcomes of their study and submit the paper for publication in a science journal. The paper is peer reviewed by scientists to validate the methods and information and, if approved, gets published so the results can be shared with the world. Cure Alzheimer’s Fund tracks published papers as one key measure of success for research grants that we funded.

In 2023, 183 peer-reviewed papers that acknowledged support from Cure Alzheimer’s Fund were published in prominent scientific journals, more than in any year before. Since our inception in 2004 through Dec. 31, 2023, the total number of published papers acknowledging CureAlz funding is 1,177. These published papers collectively have been cited by other researchers in support of their own work 97,674 times.

Here are a few select highlights from papers published resulting from CureAlz-funded research in 2023.

Scan the QR code to view a complete list of published papers or visit us online at bit.ly/2023papers

Unlocking the Brain’s Immune Gateways—The Role of Skull Channels in Neuroimmune Communication

The central nervous system (CNS), made up of the brain and spinal cord, has a specialized immune system that historically has been considered isolated from the immune system that serves the rest of the body. However, recent research has challenged this idea by demonstrating an intricate relationship between the two systems. Newly discovered skull channels connect bone marrow within the skull with the protective layers surrounding the brain, suggesting that the bone marrow contributes white blood cells to guard the CNS. bit.ly/49GmfBT

Published in *Nature Neuroscience*: “Skull bone marrow channels as immune gateways to the central nervous system”
Jonathan Kipnis, Ph.D., Washington University School of Medicine in St. Louis, and Matthias Nahrendorf, M.D., Ph.D., Massachusetts General Hospital; Harvard Medical School

Exercise and Alzheimer’s Disease: The Protective Power of Irisin*

Using a 3D cell culture model that generates Alzheimer’s disease pathology, scientists discovered that the hormone irisin, which is released from the muscles during exercise, increases the production of neprilysin in specific brain cells known as astrocytes. Upon its release from astrocytes, neprilysin breaks down amyloid beta, the protein that forms harmful plaques in AD. In these experiments, exposure to the exercise-triggered hormone irisin led to significant reduction in amyloid beta levels. bit.ly/3vbQl1T

Published in *Neuron*: “Irisin Reduces Amyloid-β by Inducing the Release of Neprilysin from Astrocytes Following Downregulation of ERK-STAT3 Signaling”
Joseph Park, Ph.D., Luisa Quinti, Ph.D., Doo Yeon Kim, Ph.D., Christiane Wrann, D.V.M, Ph.D., Rudolph Tanzi, Ph.D., and Se Hoon Choi, Ph.D., Massachusetts General Hospital; Harvard Medical School

*Research has highlighted the benefits of exercise for brain health, including for Alzheimer’s disease. The discovery of the hormone irisin and its interaction with amyloid plaques is one mechanism for exercise’s positive impact.
The Brain’s Blood Vessels Change in Alzheimer’s Disease

Changes in the blood vessels of the brain have been linked to Alzheimer’s disease (AD), and deterioration of the blood-brain barrier may be an early sign of the disease. A study profiling gene expression of the brain’s blood vessels revealed AD changes in unprecedented detail. The results provide a map to guide future therapies targeting blood-brain barrier dysfunction in Alzheimer’s.

Published in Nature Neuroscience: "Single-nucleus multiregion transcriptomic analysis of brain vasculature in Alzheimer's disease"

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

Role of T Cells in Alzheimer’s Disease Identified

With age and cell damage, microglia can shift from protective to destructive. A new study reveals that mice with loss of brain tissue caused by toxic tau, similar to that in Alzheimer’s, have more destructive T cells, generally resident in the periphery of the body, in their brains, attracted there by the brain-resident microglia. Understanding the role of these circulating immune cells in neurodegeneration may lead to new areas of therapeutic strategies.

Published in Nature: "Microglia-Mediated T Cell Infiltration Drives Neurodegeneration in Tauopathy"

Jasmin Herz, Ph.D., Jonathan Kipnis, Ph.D., Jason D. Ulinich, Ph.D., and David M. Holtzman, M.D., Washington University School of Medicine in St. Louis

Why Are Some People Resilient to Alzheimer’s Disease Pathology?

Despite high levels of amyloid beta plaques and tau tangles in their brain, some individuals never develop symptoms of dementia in their lifetime. Resilience to pathology leading to Alzheimer’s disease may be associated with reduced levels of neuroinflammation, preserving synapses and staving off neurodegeneration. This study suggests that in cases with symptoms of Alzheimer’s disease, neuroinflammation may be triggered by an unusual abundance of toxic tau oligomers in synapses that serve as an “eat me” signal to microglia and astrocytes. In contrast, resilient brains show lower levels of tau oligomers and preserved synapse numbers.

Published in JAMA Neurology: "Tau Oligomer-Containing Synapse Elimination by Microglia and Astrocytes in Alzheimer Disease"

Teresa Gomez-Isla, M.D., Massachusetts General Hospital; Harvard Medical School, and Karen E. Duff, Ph.D., University College London, England
Insights from the Aging Mouse Brain

This investigation set out to identify the molecular underpinnings of aging in the brain. It discovered that previously unsuspected regions of the brain are especially vulnerable to aging, and that two anti-aging treatments can rejuvenate the brain in unexpected ways. [bit.ly/48M91D6]

Published in Cell: “Atlas of the Aging Mouse Brain Reveals White Matter as Vulnerable Foci”
Tony Wyss-Coray, Ph.D., Stanford University

Does a Genetic Mutation Prevent Alzheimer’s Disease?

In a study exploring the interaction between genetics and Alzheimer’s disease pathology, researchers discovered how the APOE3 Christchurch (APOE3ch) mutation protects against Alzheimer’s disease. The findings showcase how APOE3ch revs up the efficiency of microglia surrounding amyloid plaques to remove aggregated tau and has the ability to prevent the spread of tau, which precedes neuronal death and dementia. [bit.ly/48JZZ9R]

Published in Cell: “APOE3ch Alters Microglial Response and Suppresses Aβ-Induced Tau Seeding and Spread”
Marco Colonna, M.D., Jason D. Ulrich, Ph.D., and David M. Holtzman, M.D., Washington University School of Medicine in St. Louis

The First Biomarker for Tau Tangles

Researchers have identified a promising biomarker, MTBR-tau243, that could transform the early detection of Alzheimer’s disease. The strong association of MTBR-tau243 with tau pathology and cognitive decline positions it as a prime candidate for diagnosing Alzheimer’s disease, tracking its advancement and evaluating tau-targeted therapies. [bit.ly/3U8xIEy]

Published in Nature Medicine: “CSF MTBR-tau243 is a specific biomarker of tau tangle pathology in Alzheimer’s disease”
David M. Holtzman, M.D., and John C. Morris, M.D., Washington University School of Medicine in St. Louis; Rik Ossenkoppele, Ph.D., Amsterdam University Medical Center, The Netherlands; Lund University, Sweden; Oskar Hansson, M.D., Ph.D., Lund University, Sweden; Randall J. Bateman, M.D., Washington University School of Medicine in St. Louis

“Funding from Cure Alzheimer’s Fund helped my lab make some important discoveries that have now launched a major area of research for us and led to several NIH grants, as well as a new drug discovery effort. So, the impact has been huge.”
—ERIK S. MUSIEK, M.D., PH.D.
Follow-on Funding

Our grants for proof-of-concept, early-stage research provide the resources to generate necessary data, leading to follow-on funding from the National Institutes of Health (NIH)/National Institute on Aging (NIA).

“Support from Cure Alzheimer’s Fund has allowed us to take risks and initiate projects quickly, without waiting years for NIH funding. It has also allowed us to build up preliminary data that has enabled us to get longer-term funding from the NIH.”

—MARC I. DIAMOND, M.D.

$64M
Total grants provided to researchers from Cure Alzheimer’s Fund 2018–2021*

$497M
Extraordinary follow-on funding from NIH/NIA

7.8x
The return of follow-on funding from NIH/NIA 2018–2023

* Latest data available
Looking Forward

This year, our 20th year of funding research, is shaping up once again to be immensely productive for Cure Alzheimer’s Fund and our researchers. There is a robust amount of grant applications being submitted by researchers with fascinating hypotheses to explore. We estimate an increase in our funding for research—with a target of $30 million—and as a result will surpass the monumental milestone of distributing $200 million in grants since our inception.

As for the science, there are several areas of research poised for breakthroughs driven by the momentum of prior discoveries, and we are looking forward to seeing what outcomes result. Two such areas of interest are the immune response and biomarkers.

Immune response research investigates the role of neuroinflammation in Alzheimer’s disease. Emerging scientific evidence suggests that in Alzheimer’s disease, the abnormal immune response in the brain is not just the work of the brain’s innate immune system. Immune cells from the periphery, part of the combined innate and adaptive immune system that functions throughout the rest of the body, have been found in the Alzheimer’s brain. Scientists are now looking to better understand this process by asking questions about how these peripheral immune cells enter the brain, why these cells are there and how they may interact with the brain’s innate immune cells to impact Alzheimer’s pathology.

Biomarkers are biological molecules that indicate the presence and potential stage of disease. There has been significant progress in biomarker research for Alzheimer’s disease, but many big questions remain. Novel biomarkers, in addition to amyloid and tau, that can measure other aspects of the disease, like neuroinflammation, neurodegeneration and synapse loss, also are actively being investigated. Predictive biomarkers that identify those at highest risk of future disease long before symptom onset will be a vital tool for making prevention strategies possible.

While we now have amyloid beta and tau biomarkers identified for Alzheimer’s disease that can be measured with positron emission tomography (PET) scans or by sampling cerebrospinal fluid, these tests can be expensive and are not always appropriate for every patient. We now also have blood tests that predict brain amyloid beta. Looking to the future, blood tests that can measure Alzheimer’s disease-related changes in various biomarkers for the disease have the potential to revolutionize diagnosis and staging of the disease.

We are energized by the work that is happening now in these areas of research and in many others, and are committed to sharing what we learn with you.

You make this work possible, and we are looking forward to another incredible year continuing our shared pursuit to gain insights and answers to the questions that will change how we address Alzheimer’s disease.
Our Researchers

This gallery features researchers who received funding in 2023, as well as the members of our Research Leadership Group and Scientific Advisory Board.

Nicola Allen, Ph.D.
Salk Institute for Biological Studies

Bérénice A. Benayoun, Ph.D.
University of Southern California

Staci Bilbo, Ph.D.
Duke University School of Medicine

Srdjan D. Antic, M.D.
University of Connecticut Health Center

Helene Benveniste, M.D., Ph.D.
Yale School of Medicine

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

Steven E. Arnold, M.D.
Massachusetts General Hospital, Harvard Medical School
New Researcher, 2023

Hebrew University of Jerusalem, Israel
New Researcher, 2023

George S. Bloom, Ph.D.
University of Virginia

Sriram Balusu, Ph.D.
KU Leuven, Belgium
New Researcher, 2023

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

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

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

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

Michael A. Bonaguidi, Ph.D.
University of Southern California

Tamir Ben-Hur, M.D., Ph.D.
Hadassah University Medical Center, Israel

Geert Jan Biessels, M.D., Ph.D.
Erasmus University Medical Center, The Netherlands

Alexandre Bonnin, Ph.D.
University of Southern California
Shane A. Liddelow, Ph.D.
New York University
Research Leadership Group

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

Derek H. Oakley, M.D., Ph.D.
Massachusetts General Hospital, Harvard Medical School
New Researcher, 2023

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

Shannon Moore, Ph.D.
University of Michigan
New Researcher, 2023

Patrick Oeckl, Ph.D.
German Center for Neurodegenerative Diseases (DZNE), Germany
New Researcher, 2023

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

John C. Morris, M.D.
Washington University School of Medicine in St. Louis
Scientific Advisory Board, Chair

Rik Ossenkoppele, Ph.D.
Amsterdam University Medical Center, The Netherlands; Lund University, Sweden

Robert C. Malenka, M.D., Ph.D.
Stanford University School of Medicine
Research Leadership Group

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

Markus Otto, M.D.
Martin-Luther-University Halle-Wittenberg, Germany
New Researcher, 2023

Edoardo Marcora, Ph.D.
Icahn School of Medicine at Mount Sinai

Erik S. Musiek, M.D., Ph.D.
Washington University School of Medicine in St. Louis

Juan Pablo Palavicini, Ph.D.
The University of Texas Health Science Center at San Antonio
New Researcher, 2023

Patrick C. May, Ph.D.
ADvantage Neuroscience Consulting LLC
Scientific Advisory Board

Liisa Myllykangas, M.D., Ph.D.
University of Helsinki, Finland

Joseph Park, Ph.D.
Massachusetts General Hospital; Harvard Medical School

Cameron McAlpine, Ph.D.
Icahn School of Medicine at Mount Sinai

Nanda Kumar Navalpur Shanmugam, Ph.D.
Massachusetts General Hospital, Harvard Medical School

Steven M. Paul, M.D.
Seaport Therapeutics
Scientific Advisory Board, Incoming Chair

Natasha McKean, Ph.D.
University of Auckland, New Zealand
New Researcher, 2023

Julia Neitzel, Ph.D.
Harvard T.H. Chan School of Public Health

Ronald C. Petersen, M.D., Ph.D.
Mayo Clinic, Rochester
Research Leadership Group

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Can (Martin) Zhang, M.D., Ph.D.
Massachusetts General Hospital; Harvard Medical School
New Researcher, 2023

Xuebing Wu, Ph.D.
Columbia University
Research Leadership Group

Riqiang Yan, Ph.D.
University of Connecticut Health Center
Research Leadership Group

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

Cheryl Wellington, Ph.D.
University of British Columbia, Canada
Research Leadership Group

Can (Martin) Zhang, M.D., Ph.D.
Massachusetts General Hospital; Harvard Medical School
New Researcher, 2023

Na Zhao, M.D., Ph.D.
Mayo Clinic, Jacksonville
New Researcher, 2023

Nancy E. Band, M.D.
University of California, San Francisco
Research Leadership Group

Yanzhuang Wang, Ph.D.
University of Michigan
New Researcher, 2023

Berislav V. Zlokovic, M.D., Ph.D.
University of Southern California
Research Leadership Group

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## 2023 Funded Research

Cure Alzheimer’s Fund spent $27.8 million to support our research program that included grants for 114 research projects across four research areas of focus. Scan the QR code to read about 2023 research projects or visit us online at [bit.ly/2023_research](http://bit.ly/2023_research).

### Project/Researcher Distribution Amount

### FOUNDATIONAL RESEARCH

#### GENETIC RISK FACTORS

<table>
<thead>
<tr>
<th>Description</th>
<th>Investigator(s)</th>
<th>Funding</th>
</tr>
</thead>
<tbody>
<tr>
<td>Analytical and Statistical Tools for Sequence Analysis for Alzheimer's Disease</td>
<td>Christoph Lange, Ph.D., Harvard T.H. Chan School of Public Health</td>
<td>$220,071</td>
</tr>
<tr>
<td>Genetic Ancestry-Specific Risk Estimation of Alzheimer's Disease in the APOE Region</td>
<td>Christoph Lange, Ph.D., Harvard T.H. Chan School of Public Health</td>
<td>$40,250</td>
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<tr>
<td>Genomic Variant Calling and Data Management for the Cure Alzheimer's Fund Alzheimer's Genome Project™</td>
<td>Winston Hide, Ph.D., Beth Israel Deaconess Medical Center; Harvard Medical School</td>
<td>$118,450</td>
</tr>
<tr>
<td>The Alzheimer's Genome Project™</td>
<td>Rudolph E. Tanzi, Ph.D., Massachusetts General Hospital; Harvard Medical School</td>
<td>$1,955,000</td>
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</table>

### BIOMARKERS, DIAGNOSTICS, AND STUDIES OF RISK AND RESILIENCE

<table>
<thead>
<tr>
<th>Description</th>
<th>Investigator(s)</th>
<th>Funding</th>
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</thead>
<tbody>
<tr>
<td>Adding Genomics and Methylomics to Personalized Disease Prediction for Alzheimer’s Disease (EPIC4AD)</td>
<td>Lars Bertram, M.D., University of Lübeck, Germany; Christina M. Lill, M.D., M.Sc., University of Münster, Germany; Imperial College London, England</td>
<td>$116,684</td>
</tr>
<tr>
<td>Cerebrospinal Fluid Neuroinflammatory Signature in Alzheimer's Disease and Related Proteopathies</td>
<td>Mathias Jucker, Ph.D., 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</td>
<td>$180,550</td>
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<tr>
<td>Characterization of Alzheimer's Disease Molecular Biomarker Profiles Throughout the Pathobiological Continuum</td>
<td>Krista L. Moulder, Ph.D., Washington University School of Medicine in St. Louis</td>
<td>$113,655</td>
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<tr>
<td>Characterization of the Longitudinal Trajectories of the Synaptic Blood Marker Beta-Synuclein During Alzheimer's Disease Pathogenesis and Improvement of the Measurement Procedure</td>
<td>Patrick Deckl, Ph.D., German Center for Neurodegenerative Diseases (DZNE), Germany; Markus Otto, M.D., Martin-Luther-University Halle-Wittenberg, Germany</td>
<td>$144,325</td>
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<tr>
<td>Identification and Validation of Plasma-Based Lipid Biomarkers for Early Alzheimer's Disease in the Unique, Primarily Hispanic, South Texas Population</td>
<td>Xianlin Han, Ph.D., The University of Texas Health Science Center at San Antonio; Juan Pablo Palavincini, 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; Bernard Fongang, Ph.D., The University of Texas Health Science Center at San Antonio</td>
<td>$201,250</td>
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</tbody>
</table>

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*Excludes research materials and scientific meeting spending.*

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**Grant Funding by Project Age (in thousands):**

- 35% New
- 46% Non-competitive Renewal
- 19% Competitive Renewal/Follow-on

**Number of New Investigators and Projects:**

<table>
<thead>
<tr>
<th>Category</th>
<th>Number</th>
<th>% of Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>New Named Investigators</td>
<td>35</td>
<td>24%</td>
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<tr>
<td>New Projects</td>
<td>43</td>
<td>38%</td>
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<tr>
<td>New Institutions</td>
<td>5</td>
<td>9%</td>
</tr>
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</table>

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**Project Age:**

- New Projects: 35% (with 19% Competitive Renewal/Follow-on and 46% Non-competitive Renewal)

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**Grant Funding:**

- New: $9,238
- Competitive Renewal/ Follow-on: $5,123
- Non-competitive Renewal: $12,070

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**Additional Information:**

- 19% Grant funding is for materials and scientific meeting spending.

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**CURE ALZHEIMER’S FUND | ANNUAL REPORT 2023**
<table>
<thead>
<tr>
<th>Project/Researcher</th>
<th>Distribution Amount</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Precision Medicine Prediction Model for Alzheimer's Disease Using Cooperative Learning Approaches for Multi-Omic Data</strong>&lt;br&gt;Christoph Lange, Ph.D., Harvard T.H. Chan School of Public Health</td>
<td>$150,000</td>
</tr>
<tr>
<td><strong>Understanding Human Brain Resilience to Alzheimer's Pathology</strong>&lt;br&gt;Teresa Gomez-Isla, M.D., Massachusetts General Hospital, Harvard Medical School</td>
<td>$300,000</td>
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<tr>
<td><strong>Utility of Blood-Based Markers for Predicting Amyloid-Related Imaging Abnormalities and Their Course in Mild Cognitive Impairment and Alzheimer's Disease Subjects Undergoing Routine Clinical Treatment with Amyloid-Directed Antibodies</strong>&lt;br&gt;Murali Doraiswamy, M.B.B.S., Duke University School of Medicine</td>
<td>$229,994</td>
</tr>
<tr>
<td><strong>BIOLOGICAL RESEARCH MATERIALS: NEW ANIMAL AND CELLULAR MODELS, AND HUMAN SAMPLES</strong>&lt;br&gt;Characterization and Validation of Two Recently Created Sheep Models of Alzheimer's Disease in Preparation for Use as a Preclinical Pharmaceutical Testing Model&lt;br&gt;Russell G. Snell, Ph.D., University of Auckland, New Zealand&lt;br&gt;Natasha McKean, Ph.D., University of Auckland, New Zealand</td>
<td>$200,933</td>
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<tr>
<td><strong>Genes to Therapies™ (G2T) Research Models and Materials</strong>&lt;br&gt;Taconic Biosciences</td>
<td>$496,283</td>
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<td><strong>Dissecting Alzheimer's Disease Phenotypes in Directly Reprogrammed Patient-Derived Neurons</strong>&lt;br&gt;Andrew S. Yoo, Ph.D., Washington University School of Medicine in St. Louis</td>
<td>$201,250</td>
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<tr>
<td><strong>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</strong>&lt;br&gt;Derek H. Oakley, M.D., Ph.D., Massachusetts General Hospital; Harvard Medical School</td>
<td>$201,250</td>
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<tr>
<td><strong>Influence of Plaque Vicinity on Microglial and Astrocyte Gene Expression; Role of Human Tau and TREM2</strong>&lt;br&gt;Frances Edwards, Ph.D., University College London, England&lt;br&gt;John Hardy, Ph.D., University College London, England</td>
<td>$172,369</td>
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<tr>
<td><strong>Multidisciplinary Phenotyping of a Novel Humanized LOAD Mouse Model</strong>&lt;br&gt;Giuseppina Tesco, M.D., Ph.D., Tufts University School of Medicine</td>
<td>$201,250</td>
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<tr>
<td><strong>TRANSLATIONAL RESEARCH</strong>&lt;br&gt;<strong>STUDIES OF NOVEL ALZHEIMER'S DISEASE GENES</strong>&lt;br&gt;<strong>ABCA7 Loss of Function in Aging and Alzheimer's Disease</strong>&lt;br&gt;Takahisa Kanekiyo, M.D., Ph.D., Mayo Clinic, Jacksonville</td>
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<td><strong>Exploring the Therapeutic Potential of Clusterin in a Preclinical Model of Alzheimer's Disease</strong>&lt;br&gt;Alban Gautier, Ph.D., University of Virginia</td>
<td>$201,250</td>
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<td><strong>Role of Ras and Rab Interactor 3 (RIN3) and Bridging Integrator 1 (BIN1) Interaction in the Neurons for Alzheimer's Disease Development</strong>&lt;br&gt;Raja Bhattacharyya, Ph.D., Massachusetts General Hospital; Harvard Medical School</td>
<td>$201,250</td>
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<td><strong>The Impact of Mutations in the Ligand-Binding Domain of CD33 on Alzheimer's Disease Pathogenesis</strong>&lt;br&gt;Ana Griciuc, Ph.D., Massachusetts General Hospital; Harvard Medical School</td>
<td>$125,000</td>
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<td><strong>STUDIES OF AMYLOID PRECURSOR PROTEIN AND AMYLOID BETA</strong>&lt;br&gt;<strong>APP Gene Dose-Mediated Dysregulation of the Endolysosomal Network Acts to Compromise Synaptic Structure and Function Leading to Alzheimer's Disease in Down Syndrome</strong>&lt;br&gt;William C. Mobley, M.D., Ph.D., University of California, San Diego</td>
<td>$230,000</td>
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<tr>
<td><strong>STUDIES OF TAU</strong>&lt;br&gt;<strong>Alzheimer's Disease Tau Consortium: How Do Soluble Tau Species Replicate</strong>&lt;br&gt;Bradley T. Hyman, M.D., Ph.D., Massachusetts General Hospital; Harvard Medical School</td>
<td>$287,169</td>
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<td><strong>Alzheimer's Disease Tau Consortium: Impact of Tau Mutations and Amyloid Beta on Tau Post-Translational Modifications and Conformation</strong>&lt;br&gt;Karen E. Duff, Ph.D., University College London, England</td>
<td>$344,993</td>
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<tr>
<td>Project/Researcher</td>
<td>Distribution Amount</td>
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<tr>
<td>Alzheimer’s Disease Tau Consortium: Role of VCP/p97 in Tau Prion Replication</td>
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<tr>
<td>Marc I. Diamond, M.D., University of Texas Southwestern Medical Center</td>
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<tr>
<td>Alzheimer’s Disease Tau Consortium: The Role of Amyloid Beta-Induced Membrane Damage in Tau Pathology</td>
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<td>Katherine Sadleir, Ph.D., Northwestern University Feinberg School of Medicine</td>
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<tr>
<td>Robert J. Vassar, Ph.D., Northwestern University Feinberg School of Medicine</td>
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<tr>
<td>Characterization of Tau Pathology Heterogeneity Across the Alzheimer’s Disease Spectrum</td>
<td>$201,250</td>
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<td>Oskar Hansson, M.D., Ph.D., Lund University, Sweden</td>
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<tr>
<td>Rik Ossenkoppele, Ph.D., Amsterdam University Medical Center, The Netherlands; Lund University, Sweden</td>
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<tr>
<td>Identifying Mediators of Tau-Mediated Neuronal Necroptosis Using an Innovative In Vivo CRISPR Screen</td>
<td>$230,000</td>
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<td>Bart De Strooper, M.D., Ph.D., KU Leuven, Belgium; University College London, England</td>
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<td>Sriram Balusu, Ph.D., KU Leuven, Belgium</td>
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<td>Multimomics Characterization of Tau Pathology Onset and Its Relationship with Amyloid in the Human Hippocampus</td>
<td>$201,250</td>
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<td>Inma Cobos, M.D., Ph.D., Stanford University</td>
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<td>Regional Variability of Pathology-Associated Properties of Tau in Posterior Cortical Atrophy</td>
<td>$172,000</td>
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<td>Bradley T. Hyman, M.D., Ph.D., Massachusetts General Hospital; Harvard Medical School</td>
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<td>John R. Dickson, M.D., Ph.D., Massachusetts General Hospital; Harvard Medical School</td>
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<td>RNA as a Determinant of Tau Seeding</td>
<td>$230,000</td>
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<td>Marc I. Diamond, M.D., University of Texas Southwestern Medical Center</td>
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<td>Targeting Microglial TSG101 for Synaptic Protection and Cognitive Enhancement in Alzheimer’s Disease</td>
<td>$172,500</td>
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<td>Seiko Ikezu, M.D., Mayo Clinic, Jacksonville</td>
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<td>Tsuneyo Ikezu, M.D., Ph.D., Mayo Clinic, Jacksonville</td>
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<td>Tau-Induced Postsynaptic Dysfunction in Tauopathy Models</td>
<td>$201,250</td>
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<td>Karin Hochrainer, Ph.D., Weill Cornell Medicine</td>
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<td>Costantino Iadecola, M.D., Weill Cornell Medicine</td>
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<td>Toxic Effects of Extracellular Tau Oligomers on Neurons</td>
<td>$192,020</td>
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<td>George S. Bloom, Ph.D., University of Virginia</td>
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<td>STUDIES OF APOLIPOPROTEIN E (APOE)</td>
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<td>Fleming APOE Consortium: APOE Genotype-Specific Effects of Human Young Plasma on Cerebrovasculature and Alzheimer’s Disease Pathology</td>
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<td>Guojun Bu, Ph.D., Hong Kong University of Science and Technology</td>
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<td>Fleming APOE Consortium: APOE4-Mediated Dysfunction of CD8 T-Cell-Microglia Crosstalk in Alzheimer’s Disease</td>
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<td>Oleg Butovsky, Ph.D., Brigham and Women’s Hospital; Harvard Medical School</td>
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<td>Fleming APOE Consortium: Modulation of Selective Neuronal Vulnerability in Alzheimer’s Disease by Apolipoprotein E</td>
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<td>Jean-Pierre Roussarie, Ph.D., Boston University</td>
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<td>Fleming APOE Consortium: Role of APOE Isoforms in Immune Responses in a Model of Tauopathy</td>
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<td>David M. Holtzman, M.D., Washington University School of Medicine in St. Louis</td>
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<td>Cellular and Molecular Studies of Apolipoprotein E Regulation of Blood-Brain Barrier, Synaptic and Neuronal Functions and Protection Strategies in Mouse Models With and Without Alzheimer’s Pathology</td>
<td>$250,000</td>
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<td>Berislav V. Zlokovic, M.D., Ph.D., University of Southern California</td>
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<td>Elucidating the Protective Effects of APOE2 in the Presence of APOE4 Gene Allele in Animal Models</td>
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<td>Na Zhao, M.D., Ph.D., Mayo Clinic, Jacksonville</td>
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<td>Yingxue Ren, Ph.D., Mayo Clinic, Jacksonville</td>
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<td>Investigating Lysosomal Mechanisms of Risk and Resilience in Alzheimer’s Disease</td>
<td>$201,250</td>
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<td>Joel Blanchard, Ph.D., Icahn School of Medicine at Mount Sinai</td>
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<td>Mitochondrial Alzheimer’s Risk Factors Control APOE Expression and Secretion</td>
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<td>Victor Faundez, M.D., Ph.D., Emory University</td>
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<td>Project/Researcher</td>
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| **Neuroproteasomes Mechanistically Connect APOE Isoforms to Endogenous Tau Aggregation**  
Kapil V. Ramachandran, Ph.D., Columbia University | $201,501            |
| **Protection Against APOE4 with Longevity-Promoting Interventions**  
Christian Pike, Ph.D., University of Southern California  
Caleb E. Finch, Ph.D., University of Southern California  
Bérénice A. Benayoun, Ph.D., University of Southern California | $234,709            |
| **STUDIES OF THE IMMUNE RESPONSE IN ALZHEIMER’S DISEASE** |                     |
| **CIRCUITS: Dissecting Microglial State Dynamics in Alzheimer’s Disease**  
Li-Huei Tsai, Ph.D., Massachusetts Institute of Technology; Broad Institute | $300,000            |
| **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            |
| **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            |
| **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 | $431,250            |
| **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 | $201,248            |
| **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            |
| **Contributions of IL-34 Signaling to Microglial Function and Alzheimer’s Pathology in Mice**  
Staci Bilbo, Ph.D., Duke University School of Medicine | $195,434            |
| **Elucidating the Role of CLEC7A in Tau-Mediated Neurodegenerative Disease**  
John R. Lukens, Ph.D., University of Virginia | $201,250            |
| **Endogenous Human Antibodies Associated with Alzheimer’s Disease**  
Charles Glabe, Ph.D., University of California, Irvine | $230,000            |
| **Extracellular ATP is a Key Factor in Promoting Alzheimer’s Disease Neuroinflammation**  
Paola Pizzo, Ph.D., University of Padova, Italy  
Francesco Di Virgilio, M.D., University of Ferrara, Italy | $150,000            |
| **Human Brain CD33 Ligand, Receptor Protein Tyrosine Phosphatase Zeta (RPTPζ)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            |
| **Investigating Bone Marrow Hematopoiesis as the Link Between Sleep Fragmentation and Vascular Inflammation in Alzheimer’s Disease**  
Cameron McAlpine, Ph.D., Icahn School of Medicine at Mount Sinai | $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 Marcara, Ph.D., Icahn School of Medicine at Mount Sinai | $201,250            |
| **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 | $201,250            |
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<tr>
<th>Project/Researcher</th>
<th>Distribution Amount</th>
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| **Neuroinflammation at the Choroid Plexus in Alzheimer's Disease**  
Maria K. Lehtinen, Ph.D., Boston Children's Hospital; Harvard Medical School  
Liisa Myllykangas, M.D., Ph.D., University of Helsinki, Finland | $201,250 |
| **Prenatal Inflammation Effects on Blood-Brain Barrier Function and Alzheimer's Disease-Related Pathologies Across the Lifespan**  
Alexandre Bonnin, Ph.D., University of Southern California | $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; Broad Institute  
Beth Stevens, Ph.D., Boston Children's Hospital; Harvard Medical School; Broad Institute | $172,550 |
| **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 |
| **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 |
| **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 |
| **T Cell Epigenetics in Alzheimer's Disease**  
David M. Gate, Ph.D., Northwestern University Feinberg School of Medicine | $172,500 |
| **Tau and Amyloid Beta are Innate Immune Antimicrobial Peptides in the Brain**  
William Eimer, Ph.D., Massachusetts General Hospital; Harvard Medical School | $172,500 |
| **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 | $172,500 |
| **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 |
| **To Elucidate the Role of Memory T Cells as a Determinant of Age-Related Inflammation in Alzheimer's Disease**  
Susan M. Kaech, Ph.D., Salk Institute for Biological Studies | $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 |
| **Understanding the Role of Natural Amyloid Beta-Specific B Cell Responses in Alzheimer's Disease Progression**  
Marco Colonna, M.D., Washington University School of Medicine in St. Louis | $172,500 |
| **STUDIES OF ALTERNATIVE NEURODEGENERATIVE PATHWAYS** | |
| **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: 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 |
| **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  
Allen R. Tannenbaum, Ph.D., State University of New York at Stony Brook | $204,081 |
| **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 |
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<td><strong>Brain Entry and Exit Consortium: Identifying the Blood-Brain Barrier Changes During Alzheimer's Disease</strong>&lt;br&gt;Richard Daneman, Ph.D., University of California, San Diego</td>
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<td><strong>Microbiome Consortium: Harnessing Diet-Microbe Interactions to Prevent Alzheimer's Disease Pathogenesis</strong>&lt;br&gt;Laura M. Cox, Ph.D., Brigham and Women's Hospital; Harvard Medical School</td>
<td>$287,500</td>
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<td><strong>Microbiome Consortium: Interaction of the Microbiome with Astrocytes and Amyloid Pathology</strong>&lt;br&gt;Robert J. Vassar, Ph.D., Northwestern University Feinberg School of Medicine</td>
<td>$345,000</td>
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<td><strong>Microbiome Consortium: Profiling of Human Brain and Gut Microbiomes in Alzheimer's Disease</strong>&lt;br&gt;Rudolph E. Tanzi, Ph.D., Massachusetts General Hospital; Harvard Medical School&lt;br&gt;Nanda Kumar Navalpur Shanmugam, Ph.D., Massachusetts General Hospital; Harvard Medical School</td>
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<td><strong>Microbiome Consortium: Temporal Relationships Between Gut Dysbiosis, Brain Amyloid Beta Metabolism and Microglia Cell Activation Following Antibiotic Treatment</strong>&lt;br&gt;Sangram S. Sisodia, Ph.D., University of Chicago</td>
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<td><strong>Microbiome Consortium: The Role of Gut Microbial Metabolism in Tau-Mediated Neurodegeneration</strong>&lt;br&gt;David M. Holtzman, M.D., Washington University School of Medicine in St. Louis</td>
<td>$287,500</td>
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<td><strong>A Multimodality Study on the Lipid Molecular Basis of Obesity and Its Roles in Regulating Alzheimer's Pathogenesis for Developing Potential Targeted Interventions</strong>&lt;br&gt;Stephen T.C. Wong, Ph.D., Houston Methodist Research Institute; Weill Cornell Medicine</td>
<td>$201,199</td>
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<td><strong>Air Pollution and Alzheimer's Disease Risk Interact with Premature Aging of Neural Stem Cells and Apolipoprotein E Alleles</strong>&lt;br&gt;Caleb E. Finch, Ph.D., University of Southern California&lt;br&gt;Michael A. Bonaguidi, Ph.D., University of Southern California</td>
<td>$301,069</td>
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<td><strong>Alzheimer's Disease Pathophysiology Alters the Level of Electrical and Chemical Synapse Coupling in the Network of GABAergic PV+ Interneurons Early in Disease Course</strong>&lt;br&gt;Srdjan D. Antic, M.D., University of Connecticut Health Center&lt;br&gt;Riqiang Yan, Ph.D., University of Connecticut Health Center</td>
<td>$230,000</td>
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<td><strong>Characterizing Gut Bacteriome-Mycobiome Synergy in Correlation to Amylin-Amyloid Beta Antimicrobial Synergy in Alzheimer's Disease (AD) in AD Mouse Models</strong>&lt;br&gt;Deepak Kumar Vijaya Kumar, Ph.D., Massachusetts General Hospital; Harvard Medical School</td>
<td>$201,250</td>
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<td><strong>Circadian Desynchrony, Glial Dysfunction and Alzheimer's Disease Pathogenesis</strong>&lt;br&gt;Erik S. Musiek, M.D., Ph.D., Washington University School of Medicine in St. Louis</td>
<td>$198,994</td>
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<td><strong>Deciphering and Restoring Computational Setpoints in Alzheimer's Disease Through Sleep-Enhanced Network Homeostasis</strong>&lt;br&gt;Keith B. Hengen, Ph.D., Washington University in St. Louis</td>
<td>$189,902</td>
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<td><strong>Decoding Microbial Products Modulating Alzheimer's Disease—Toward Precision Postbiotics Treatment</strong>&lt;br&gt;Eran Elinav, M.D., Ph.D., Weizmann Institute of Science, Israel; DKFZ, Germany</td>
<td>$201,250</td>
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<td><strong>Disentangling the Role of Intracranial Arteriosclerosis in Alzheimer's Disease</strong>&lt;br&gt;Daniel Bos, M.D., Ph.D., Erasmus University Medical Center, The Netherlands&lt;br&gt;Meike Vernooij, M.D., Ph.D., Erasmus University Medical Center, The Netherlands&lt;br&gt;Frank J. Wolters, M.D., Ph.D., Erasmus University Medical Center, The Netherlands&lt;br&gt;Geert Jan Biessels, M.D., Ph.D., Erasmus University Medical Center, The Netherlands&lt;br&gt;Julia Neitzel, Ph.D., Harvard T.H. Chan School of Public Health</td>
<td>$167,207</td>
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<td><strong>Effect of APOE Genotype in a Novel Rat Model of Cerebral Amyloid Angiopathy</strong>&lt;br&gt;William Van Nostrand, Ph.D., The University of Rhode Island</td>
<td>$201,250</td>
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<td><strong>Evaluating TMEM106B Accumulation in Alzheimer's Disease</strong>&lt;br&gt;Leonard Petrucelli, Ph.D., Mayo Clinic, Jacksonville&lt;br&gt;Casey N. Cook, Ph.D., Mayo Clinic, Jacksonville</td>
<td>$201,250</td>
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<td>Functional Changes to Cerebrospinal Fluid Immune Cells Resulting from Bacillus Calmette-Guérin (BCG) Vaccination in Older Adults With and Without Alzheimer's Disease</td>
<td>$258,750</td>
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| Marc Weinberg, M.D., Ph.D., Massachusetts General Hospital; Harvard Medical School  
Steven E. Arnold, M.D., Massachusetts General Hospital; Harvard Medical School |                     |
| In Vivo Models for Golgi Fragmentation and the Molecular Pathogenesis of Alzheimer's Disease | $230,000            |
| Samuel E. Gandy, M.D., Ph.D., Icahn School of Medicine at Mount Sinai  
Yanzhuang Wang, Ph.D., University of Michigan |                     |
| Morphological, Electrophysiological and Transcriptional Characterization of Single Neurons from Resilient and Susceptible Models of Human Alzheimer's Disease | $201,250            |
| Catherine Kaczorowski, Ph.D., University of Michigan  
Shannon Moore, Ph.D., University of Michigan |                     |
| Neuronal Mechanisms Driving Synapse Loss in Alzheimer's Disease | $201,250            |
| Martin Kampmann, Ph.D., University of California, San Francisco |                     |
| Neuroprotective Effects of the Exercise Hormone irisin in Alzheimer's Disease | $345,000            |
| Se Hoon Choi, Ph.D., Massachusetts General Hospital; Harvard Medical School  
Christiane Wrann, D.V.M, Ph.D., Massachusetts General Hospital; Harvard Medical School |                     |
| Noncoding Translation Feedback Loop in Alzheimer's Disease | $201,250            |
| Xuebing Wu, Ph.D., Columbia University |                     |
| Oligodendroglial Dynamics and Myelination in Alzheimer's Disease | $198,751            |
| Erin M. Gibson, Ph.D., Stanford University |                     |
| Restore Meningeal Lymphatic Drainage to Alleviate White Matter Damage and Cerebral Amyloid Angiopathy in a Model of Alzheimer's Disease | $201,250            |
| Sandro Da Mesquita, Ph.D., Mayo Clinic, Jacksonville |                     |
| Scaling the Divide in Alzheimer's Disease: An Integrated Molecular, Cellular and Network-Level Study | $191,624            |
| Marc Aurel Busche, M.D., Ph.D., University College London, England  
Samuel Harris, Ph.D., University College London, England |                     |
| Stress and Neurovascular-Immune Networks in Alzheimer's Disease | $172,500            |
| Scott J. Russo, Ph.D., Icahn School of Medicine at Mount Sinai  
Wolfram C. Poller, M.D., Icahn School of Medicine at Mount Sinai |                     |
| Targeting the Microbiome and Innate Immunity in Alzheimer's Disease | $201,250            |
| 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 |                     |
| Understanding the Mechanism Underlying Vaccination for Alzheimer's Disease | $115,805            |
| 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 |                     |
| DRUG DISCOVERY |
| Identification and Development of CD33 Inhibitors and Pre-RNA Splicing Modulators | $201,250            |
| Subhash Sinha, Ph.D., Weill Cornell Medicine |                     |
| Validation and Characterization of Compounds Modulating Neuroinflammation and Amyloid Beta Uptake in Microglial Cells | $201,250            |
| Ana Griciuc, Ph.D., Massachusetts General Hospital; Harvard Medical School  
Luisa Quinti, Ph.D., Massachusetts General Hospital; Harvard Medical School |                     |
### PRECLINICAL AND CLINICAL DRUG DEVELOPMENT

#### PRECLINICAL DRUG DEVELOPMENT

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<td><strong>Characterization of CNS-Penetrant HDAC11-Selective Inhibitors in Alzheimer's Disease</strong>&lt;br&gt;Can (Martin) Zhang, M.D., Ph.D., Massachusetts General Hospital; Harvard Medical School&lt;br&gt;Changning Wang, Ph.D., Massachusetts General Hospital; Harvard Medical School</td>
<td>$201,250</td>
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<td><strong>Development of Human cGAS Inhibitors to Treat Alzheimer's Disease</strong>&lt;br&gt;Li Gan, Ph.D., Weill Cornell Medicine&lt;br&gt;Subhash Sinha, Ph.D., Weill Cornell Medicine</td>
<td>$250,000</td>
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<td><strong>Interrogating Levetiracetam's Impact on Amyloid Pathology and Presynaptic Proteostasis in Knock-In Mouse Models with Humanized Amyloid Beta</strong>&lt;br&gt;Jeffrey Savas, Ph.D., Northwestern University</td>
<td>$136,827</td>
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<tr>
<td><strong>Role of Stabilization of MAMs and MAM-Associated Palmitoylated APP (MAM-palAPP) in Alzheimer’s Disease</strong>&lt;br&gt;Raja Bhattacharyya, Ph.D., Massachusetts General Hospital; Harvard Medical School</td>
<td>$201,250</td>
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<td><strong>Targeting Neuroinflammation with Nasal Administration of Anti-CD3 Monoclonal Antibody to Treat Alzheimer’s Disease</strong>&lt;br&gt;Rafael M. Rezende, Ph.D., Brigham and Women's Hospital; Harvard Medical School</td>
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#### CLINICAL TRIAL DESIGN

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<td><strong>A Proposal to Evaluate the Effect of Bacillus Calmette-Guérin Vaccination on Alzheimer’s Disease Development</strong>&lt;br&gt;Tamir Ben-Hur, M.D., Ph.D., Hadassah University Medical Center, Israel&lt;br&gt;Herve Bercovier, D.V.M., M.Sc., Hebrew University of Jerusalem, Israel</td>
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#### OTHER

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<td><strong>General Scientific Support</strong>&lt;br&gt;Wilma Wasco, Ph.D., Massachusetts General Hospital; Harvard Medical School</td>
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<td><strong>Scientific Meeting Support</strong></td>
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# Ongoing Research Projects

The research projects listed here were ongoing and active in 2023, having received funding from Cure Alzheimer’s Fund in a previous year.

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<td><strong>GENETIC RISK FACTORS</strong></td>
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<tr>
<td>Analytical and Statistical Tools for Sequence Analysis for Alzheimer’s Disease</td>
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<td>Christoph Lange, Ph.D., Harvard T.H. Chan School of Public Health</td>
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<td><strong>BIONARKERS, DIAGNOSTICS, AND STUDIES OF RISK AND RESILIENCE</strong></td>
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<tr>
<td>Harnessing Big Data to Understand Alzheimer’s Disease Risk</td>
<td>$172,500</td>
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<td>Brad A. Racette, M.D., Barrow Neurological Institute; Susan Searles Nielsen, Ph.D., and Alejandra Camacho-Soto, M.D., M.P.H.S., Washington University School of Medicine in St. Louis</td>
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<tr>
<td>Henry L. Paulson, M.D., Ph.D., Bruno Giordani, Ph.D., and Benjamin M. Hampstead, Ph.D., ABPP/CN, University of Michigan</td>
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<td>Personalized Disease Prediction for Alzheimer’s Disease Using Proteome Profiling: The EPIC4AD Study</td>
<td>$541,897</td>
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<td>Christina M. Lill, M.D., M.Sc., University of Münster, Germany; Imperial College London, England Lars Bertram, M.D., University of Lübeck, Germany</td>
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<td>Sex Differences in Alzheimer’s Disease Progression: Framingham Heart Study</td>
<td>$199,162</td>
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<td>Murali Doraiswamy, M.B.B.S., Duke University School of Medicine</td>
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<tr>
<td>Stable Isotope Labeling and Quantitative Mass Spectrometry Imaging of Alzheimer’s Disease Pathology in Human Brain</td>
<td>$150,000</td>
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<td>Katherine Schwetye, M.D., Ph.D., Washington University School of Medicine in St. Louis</td>
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<td><strong>BIOLOGICAL RESEARCH MATERIALS: NEW ANIMAL AND CELLULAR MODELS, AND HUMAN SAMPLES</strong></td>
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<td>Creation of a Fibroblast/iPS Cell Bank to Facilitate Peripheral/Brain Comparisons, and Allow Molecular Investigations into Molecular Mechanisms Underlying Differences in Disease Aggressiveness</td>
<td>$250,000</td>
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<td>Bradley T. Hyman, M.D., Ph.D., Massachusetts General Hospital; Harvard Medical School</td>
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<td>Development of a Multicellular Brain Model to Study Brain-Vascular-Peripheral Immune Cells Crosstalk in Alzheimer’s Disease</td>
<td>$172,500</td>
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<td>Mehd Jorfi, Ph.D., Joseph Park, Ph.D., and Rudolph E. Tanzi, Ph.D., Massachusetts General Hospital; Harvard Medical School</td>
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<td>Neuronal Subtype-Specific Modeling of Alzheimer’s Disease by Direct Neuronal Reprogramming of Patient Fibroblasts</td>
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<td>Andrew S. Yoo, Ph.D., Washington University School of Medicine in St. Louis</td>
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<td><strong>EPIGENETIC FACTORS</strong></td>
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<td>CIRCUITS: A Unified Approach to Actionable Alzheimer’s Disease Signatures</td>
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<td>Winston Hide, Ph.D., Beth Israel Deaconess Medical Center; Harvard Medical School</td>
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<td>CIRCUITS: Characterizing Epigenetic Biomarkers of Human Cognitive Aging</td>
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<td>Lars Bertram, M.D., University of Lübeck, Germany</td>
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<td>CIRCUITS: Consortium to Infer Regulatory Circuits and Uncover Innovative Therapeutic Strategies—Production Group</td>
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<td>Manolis Kellis, Ph.D., and Li-Huei Tsai, Ph.D., Massachusetts Institute of Technology; Broad Institute</td>
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<td>CIRCUITS: Impact of Genetic, Epigenetic and Cellular Variants on Alzheimer’s Disease Pathology</td>
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<td>Rudolf Jaenisch, M.D., Whitehead Institute, Massachusetts Institute of Technology Joseph R. Ecker, Ph.D., Salk Institute for Biological Studies</td>
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<td>CIRCUITS: Interpreting Alzheimer’s Disease-Associated Genetic Variation at Enhancer Regions</td>
<td>$200,000</td>
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<td>Andreas R. Pfenning, Ph.D., Carnegie Mellon University</td>
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## TRANSLATIONAL RESEARCH

### STUDIES OF NOVEL ALZHEIMER’S DISEASE GENES

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<tr>
<td><strong>ABCA7 Loss of Function in Aging and Alzheimer’s Disease</strong></td>
<td>$172,500</td>
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<tr>
<td>Takahisa Kanekiyo, M.D., Ph.D., Mayo Clinic, Jacksonville</td>
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<td>Guojun Bu, Ph.D., Hong Kong University of Science and Technology</td>
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<td><strong>Dissecting the Modulatory Roles of Interleukin-17 Receptor D in Alzheimer’s Disease</strong></td>
<td>$201,250</td>
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<td>Jun Huh, Ph.D., Harvard Medical School</td>
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<td><strong>Functional Basis for Novel Protein Kinase C-eta K46R Mutation in Alzheimer’s Disease</strong></td>
<td>$172,500</td>
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<tr>
<td>Alexandra C. Newton, Ph.D., University of California, San Diego</td>
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<td><strong>In Vivo Characterization of a Loss-of-Function GGA3 Rare Variant Associated with Alzheimer’s Disease</strong></td>
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<td>Giuseppina Tesco, M.D., Ph.D., Tufts University School of Medicine</td>
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<tr>
<td><strong>Single Nucleus RNA Sequencing Analysis of ACE1 R1284Q Knockin Mice</strong></td>
<td>$246,804</td>
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<td>Robert J. Vassar, Ph.D., David M. Gate, Ph.D., and Leah Cuddy, Ph.D., Northwestern University Feinberg School of Medicine</td>
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<td><strong>Understanding, and Mimicking, the Biological Effects of the Phospholipase C-gamma-2 P522R Variant That Protect Against Alzheimer’s Disease</strong></td>
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<td>Rik van der Kant, Ph.D., Amsterdam University Medical Center, The Netherlands</td>
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### STUDIES OF AMYLOID PRECURSOR PROTEIN AND AMYLOID BETA

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<tr>
<td><strong>Effects of Depalmitoylation and ACAT Inhibition on Axonal Amyloid Beta Generation via MAM-Associated palAPP</strong></td>
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<td>Raja Bhattacharyya, Ph.D., and Rudolph E. Tanzi, Ph.D., Massachusetts General Hospital; Harvard Medical School</td>
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<td><strong>SFRP1 as a Therapeutic Target and Diagnostic/Prognostic Factor In Alzheimer’s Disease</strong></td>
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<td>Paola Bovolenta, Ph.D., Universidad Autónoma de Madrid, Spain</td>
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<tr>
<td><strong>Structural Mimicry in Microbial and Antimicrobial Amyloids Connected to Neurodegenerative Diseases</strong></td>
<td>$200,760</td>
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<tr>
<td>Meytal Landau, B. Pharm., M.Sc., Ph.D., Technion, Israel Institute of Technology; Deutsches Elektronen-Synchrotron (DESY), Germany</td>
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### STUDIES OF TAU

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<tr>
<td><strong>Alzheimer’s Disease Tau Consortium: Deep Mass Spectrometry Profiling of Tau Aggregates in Alzheimer’s Disease and Other Tauopathies</strong></td>
<td>$287,500</td>
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<tr>
<td>Henrik Zetterberg, M.D., Ph.D., and Gunnar Brinkmalm, Ph.D., University of Gothenburg, Sweden</td>
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<td><strong>Investigating the Role of Tau Protein in Neuronal Senescence Induction and Maintenance</strong></td>
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<td>Miranda E. Orr, Ph.D., Wake Forest University School of Medicine</td>
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<td><strong>Mechanisms of Tau Propagation Across the Plasma Membrane</strong></td>
<td>$250,000</td>
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<td>Marc I. Diamond, M.D., University of Texas Southwestern Medical Center</td>
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<td><strong>Reversal of Tau Pathology by an Adenosine A1 Receptor Antagonist</strong></td>
<td>$287,500</td>
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<td>Eckhard Mandelkow, Ph.D., Eva-Maria Mandelkow, M.D., Ph.D., and Anja Schneider, M.D., German Center for Neurodegenerative Diseases (DZNE), Germany</td>
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<td><strong>Targeting Tauopathies with Antisense Oligonucleotides to Synaptogyrin-3</strong></td>
<td>$215,625</td>
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<td>Patrik Verstreken, Ph.D., VIB-KU Leuven, Belgium</td>
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<td><strong>Using Long-Read Sequencing to Investigate the MAPT Locus and Transcripts in Neurodegeneration</strong></td>
<td>$201,250</td>
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<td>John Hardy, Ph.D., University College London, England</td>
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## CURE ALZHEIMER’S FUND

**ANNUAL REPORT 2023**

### Project/Researcher Distribution Amount

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<tr>
<th>Study Title</th>
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<td><strong>STUDIES OF APOLIPROTEIN E (APOE)</strong></td>
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<tr>
<td>Fleming APOE Consortium: Assessing the Added Diagnostic Value of Peripheral Apolipoprotein E Protein Levels in Current Blood-Based Biomarker Assays for Central Nervous System Amyloidosis</td>
<td>Randall J. Bateman, M.D., Washington University School of Medicine in St. Louis</td>
<td>$252,077</td>
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<td>Fleming APOE Consortium: Effect of Cholesteryl Ester Transfer Protein Activity on Amyloid and Cerebrovascular Pathologies in Animal Models of Alzheimer’s Disease</td>
<td>Cheryl Wellington, Ph.D., University of British Columbia, Canada</td>
<td>$287,500</td>
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<tr>
<td>Apolipoprotein E and Immunometabolism in Alzheimer’s Disease</td>
<td>Lance A. Johnson, Ph.D., Ramon Sun, Ph.D., and Josh Morganti, Ph.D., University of Kentucky College of Medicine</td>
<td>$172,500</td>
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<tr>
<td>Establishing the Molecular and Cellular Mechanisms and Biomarkers of APOE4-Mediated Susceptibility to Tau-Related Cognitive Impairments</td>
<td>Joel Blanchard, Ph.D., Icahn School of Medicine at Mount Sinai</td>
<td>$172,500</td>
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<tr>
<td>Sex Matters: Understanding the Influence of Sex and Apolipoprotein E (APOE) Genotype on Hippocampal Plasticity and Cognition</td>
<td>Liisa Galea, Ph.D., University of British Columbia, Canada</td>
<td>$170,200</td>
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<td><strong>STUDIES OF THE IMMUNE RESPONSE IN ALZHEIMER’S DISEASE</strong></td>
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<td>Neuroimmune Consortium: Assessing the Links Between the MS4A Risk Genes, Microglia and Alzheimer’s Disease</td>
<td>Sandeep Robert Datta, M.D., Ph.D., Harvard Medical School</td>
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<td>Neuroimmune Consortium: Biomarker Tool Development</td>
<td>Jacob Hooker, Ph.D., Massachusetts General Hospital; Harvard Medical School</td>
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<td>Neuroimmune Consortium: Examining the Role of Human Microglia in the Transition Between Parenchymal and Vascular Beta-Amyloid Pathology</td>
<td>Mathew Blurton-Jones, Ph.D., University of California, Irvine</td>
<td>$250,000</td>
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<td>Neuroimmune Consortium: Investigation of Alzheimer’s Disease Risk Alleles in Astrocytes—Focus on Cholesterol Transport and Microglia Interactions</td>
<td>Shane A. Liddelow, Ph.D., New York University</td>
<td>$115,000</td>
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<td>Neuroimmune Consortium: Leveraging Enhancer Landscapes to Decode Alzheimer’s Disease Risk Alleles in Microglia</td>
<td>Christopher K. Glass, M.D., Ph.D., University of California, San Diego</td>
<td>$250,000</td>
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<td>Neuroimmune Consortium: Understanding the Consequences of Noncoding Alzheimer’s Disease Risk Alleles on Microglia Function</td>
<td>Beth Stevens, Ph.D., Boston Children’s Hospital; Harvard Medical School; Broad Institute</td>
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<td>Elucidating the Role of Soluble Epoxide Hydrolase and Arachidonic Acid Metabolism in Neuroinflammation and Alzheimer’s Disease</td>
<td>Hui Zheng, Ph.D., Baylor College of Medicine</td>
<td>$167,637</td>
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<td>Role of Checkpoint Molecule TIM-3 in Regulating Microglia in Alzheimer’s Disease</td>
<td>Vijay K. Kuchroo, D.V.M., Ph.D., Brigham and Women’s Hospital; Harvard Medical School</td>
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<td>Role of Microglia in Degradation and Trimming of Alzheimer’s Amyloid Beta</td>
<td>Frederick R. Maxfield, Ph.D., Weil Cornell Medical College</td>
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<td>Role of SPARC in Immunometabolic Control of Age-Related Inflammation</td>
<td>Vishwa Deep Dixit, D.V.M., Ph.D., Yale School of Medicine</td>
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<td>Targeting a Master Innate Immune Adaptor Molecule in Alzheimer’s Disease</td>
<td>John R. Lukens, Ph.D., University of Virginia School of Medicine</td>
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<td>Targeting Reactive Astrocytes for Therapeutic Intervention of Alzheimer’s Disease</td>
<td>Gilbert Gallardo, Ph.D., Washington University School of Medicine in St. Louis</td>
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## ONGOING RESEARCH PROJECTS (CONTINUED)

<table>
<thead>
<tr>
<th>Project/Researcher</th>
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| **The Neuroprotective Gial Barrier: A Multicellular Reaction with Therapeutic Potential in Alzheimer's Disease**  
Jaime Grutzendler, M.D., Yale School of Medicine | $172,500            |
| **The Role of Astrocyte-Derived Toxic Lipids Mediating Degeneration in Alzheimer's Disease**  
Shane A. Liddelow, Ph.D., New York University | $174,883            |
| **VGF-Derived Peptide Therapy for Alzheimer's Disease: Studies of Mouse and Human TLQP-21 and its Receptor, C3aR1**  
Michele E. Ehrlich, M.D., and Stephen R. Salton, M.D., Ph.D., Icahn School of Medicine at Mount Sinai | $172,500            |
| **STUDIES OF ALTERNATIVE NEURODEGENERATIVE PATHWAYS** |                       |
| **Cellular Vulnerability to Aging in Alzheimer's Disease**  
Mathieu Bourdenx, Ph.D., and Karen E. Duff, Ph.D., University College London, England | $230,000            |
| **Characterizing Gut Microbiome Synergy With Emphasis on Mycobiome and Its Impact on Alzheimer's Disease (AD) Pathology in AD Mouse Models**  
Deepak Kumar Vijaya Kumar, Ph.D, Nanda Kumar Navalpur Shanmugam, Ph.D., William Eimer, Ph.D., and Rudolph E. Tanzi, Ph.D., Massachusetts General Hospital; Harvard Medical School | $250,000            |
| **Circadian Perturbations of the Vasculome and Microgiome in Alzheimer's Disease**  
Eng H. Lo, Ph.D., Massachusetts General Hospital; Harvard Medical School | $200,417            |
| **Gut Microbiota, Endothelial Dysfunction and Tau-Mediated Cognitive Impairment**  
Giuseppe Faraco, M.D., Ph.D., and Costantino Iadecola, M.D., Weill Cornell Medicine | $172,500            |
| **Harnessing Meningeal Lymphatics and Immunity to Alleviate APOE4-Induced Brain Dysfunction**  
Sandro Da Mesquita, Ph.D., Mayo Clinic, Jacksonville | $172,500            |
| **Identifying the Sex-Specific Roles of the Gut Microbiome-Brain Axis in a Mouse Model of Amyloid Beta Amyloidosis**  
Sangram S. Sisodia, Ph.D., University of Chicago | $210,871            |
| **Immunotherapies Targeting the Microbiota to Prevent Cognitive Decline in Alzheimer's Disease**  
Gerald B. Pier, Ph.D., Colette Cywes-Bentley, Ph.D., and Cynthia A. Lemere, Ph.D., Brigham and Women's Hospital; Harvard Medical School | $183,562            |
| **Molecular Signatures of APOE-Mediated Blood-Brain Barrier Dysfunction Causing Neuronal and Synaptic Dysfunction**  
Berislav V. Zlokovic, M.D., Ph.D., University of Southern California | $250,000            |
| **Neural Synaptic Circuit Changes During Alzheimer's Disease Progression**  
Huizhong W. Tao, Ph.D., University of Southern California | $172,500            |
| **Neuroinflammation Contributions to Alzheimer's Disease: Role of the Choroid Plexus**  
Maria K. Lehtinen, Ph.D., Boston Children's Hospital; Harvard Medical School; and Lisa Myllykangas, M.D., Ph.D., University of Helsinki, Finland | $172,500            |
| **Role of the Circulating Exerkine GPLD1 in Ameliorating Alzheimer's Disease Pathology**  
Saul Villeda, B.S., Ph.D., University of California, San Francisco | $201,250            |
| **Temporal Relationships Between Gut Dysbiosis and Microglia Cell Activation Following Antibiotic Treatment**  
Sangram S. Sisodia, Ph.D., University of Chicago | $229,033            |
| **Turning Up Mitophagy to Blunt Alzheimer's Tau Pathologies**  
Evandro F. Fang, Ph.D., University of Oslo, Norway | $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            |
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<td><strong>DRUG DISCOVERY</strong></td>
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<td><strong>DRUG SCREENING AND LEAD DRUG EVALUATION PROJECTS</strong></td>
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<td>Alzheimer's Disease Drug Discovery and Development Consortium: Blocking Synaptotoxicity in Alzheimer's Three-Dimensional Models Weiming Xia, Ph.D., Boston University</td>
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<tr>
<td>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., and Luisa Quinti, Ph.D., Massachusetts General Hospital; Harvard Medical School</td>
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<td>Alzheimer's Disease Drug Discovery and Development Consortium: Modulating CD33 Function and Neuroinflammation as a Therapeutic Approach for Alzheimer's Disease Ana Griciuc, Ph.D., Massachusetts General Hospital; Harvard Medical School</td>
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<td>Alzheimer's Disease Drug Discovery and Development Consortium: Uncovering the Molecular Mechanism of Selected Drug Candidates Derived From Systematic Alzheimer's Drug Repositioning Stephen T.C. Wong, Ph.D., Houston Methodist Research Institute; Weill Cornell Medicine</td>
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<td>A Transcriptional Rejuvenation Signature for Alzheimer's Disease Tony Wyss-Coray, Ph.D., Stanford University</td>
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<td>Identification of CD33 Antagonists Subhash Sinha, Ph.D., Weill Cornell Medicine</td>
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<td>Small Molecule Activators of PLC-gamma-2 as Novel Therapeutics for Alzheimer's Disease Qisheng Zhang, Ph.D., John Sondek, Ph.D., and Kenneth Pearce, Ph.D., University of North Carolina at Chapel Hill</td>
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<td>Stimulating Synaptic Proteasome Activity for the Treatment of Alzheimer's Disease Hermann Steller, Ph.D., The Rockefeller University</td>
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<td><strong>DRUG DELIVERY AND ENABLING TECHNOLOGIES</strong></td>
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<td>Novel Entry Routes for Therapeutic Biologicals to the Brain Maarten Dewilde, Ph.D., KU Leuven, Belgium, and Bart De Strooper, M.D., Ph.D., KU Leuven, Belgium; University College London, England</td>
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<tr>
<td><strong>PRECLINICAL AND CLINICAL DRUG DEVELOPMENT</strong></td>
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<td><strong>PRECLINICAL DRUG DEVELOPMENT</strong></td>
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<td>Combined Hormone Therapy as a Novel Treatment for Alzheimer's Disease in the Face of a Metabolic Challenge: Influence of Sex and Genotype Lisa Galea, Ph.D., and Annie Ciernia, Ph.D., University of British Columbia, Canada</td>
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<td>Continuing Studies of the Effects of GSM 776890 Administration on Amyloid Species and Microgliosis in Older Alzheimer's Model Mice Kevin Rynearson, M.S., Ph.D., University of California, San Diego</td>
<td>$291,374</td>
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<td><strong>CLINICAL TRIAL DESIGN</strong></td>
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<td>Application of Machine Learning Methods in Alzheimer's Disease Clinical Trials Ali Ezzati, M.D., University of California, Irvine, and Richard B. Lipton, M.D., Albert Einstein Medical College</td>
<td>$100,000</td>
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Working Together Toward a Cure

Alzheimer’s disease has touched millions of people—and every single person who has been affected has a unique story to tell. It is through sharing our experiences that we understand the true impact of this disease on individuals, their families and friends. By working together to solve the problem through research, we will move ever closer to a cure.

FRED RYERSE AND THE RYERSE FAMILY

“My wife Judi and I were married for 58 years. We supported many charities throughout our lives, but when Judi was diagnosed with Alzheimer’s in 2012, we started giving to Alzheimer’s research. We asked our five kids and 16 grandkids if they would be OK with forgoing Christmas presents so we could contribute even more. Not only were there no objections, but many of our extended family members chose to donate as well. Since then, our family giving has grown exponentially, including matching grants and ongoing contributions since Judi’s passing in 2019. Our support of CureAlz research is given out of love. We hope to move more people to support research for a cure.”

BOB WELLMAN

“I chose to support CureAlz because I know that every penny I donate will be disbursed in grants and not used for operating expenses. That’s unique. Moreover, CureAlz is transparent about where the money goes. The caliber of scientists supported is top tier, and the research is vitally important and urgently needed.

“I donate in honor of my wife, who died of Alzheimer’s at age 77 after a decade of declining function. Mary was a brilliant psychologist, educator, clinician and researcher. She was also warm, caring, fiercely loyal to friends, and both subtly and overtly funny. Watching those capacities fade far too early prompts me to support efforts to ensure that others might not need to endure such losses.

“I’m supporting CureAlz in two ways: (1) direct annual contributions of the required minimum distribution from my IRA, and (2) beneficiary designation from retirement accounts. This is, by far, my most important charitable contribution. I urge anyone who has been personally touched by Alzheimer’s to support CureAlz. You’ll be glad you did!”

Judi and Fred Ryerse

Mary and Bob Wellman

Judi and Fred Ryerse

46 | ANNUAL REPORT 2023 | WORKING TOGETHER TOWARD A CURE
RUNNING 4 ANSWERS

“Over $500,000 in donations. 15 years. Alzheimer’s affected my family like it has thousands of others. My mom started having Alzheimer’s symptoms around age 54, a year younger than I am now. In an effort to ‘do something,’ ‘Running 4 Answers, a Race Against Alzheimer’s’ was created. That’s the thing about Alzheimer’s—there isn’t much ‘to do.’ There aren’t a lot of appointments or treatments to try. Alzheimer’s leaves you watching your loved one fade away piece by piece. Slowly. This 5K race and 2-mile walk gave not just me, but a whole community, a way to show support, [and] raise money and awareness of this sad, devastating disease. I can’t thank Cure Alzheimer’s Fund enough for their support and unending dedication to finding a way to slow, stop or reverse Alzheimer’s. While this event may have ended in 2024, my work with them will continue.”

Our deepest thanks to Carolyn, the hundreds of volunteers and thousands of runners and walkers who have made Running 4 Answers such an amazing event for 15 glorious years. We are forever grateful.

TY AND SARAH NEWELL

“Grandparents on both sides of our family were touched with the disease. The gradual loss of memory, independence and identity left a mark on our families. Witnessing the pain our parents experienced, compounded with our fear of how our own parents may be impacted by the disease, pushed us to want to find ways to help.

“We decided that putting dollars directly toward research efforts to combat the condition would help drive measurable change and provide us with a front-row seat to the latest academia and industry knowledge.

“While a cure would be the ultimate ‘win,’ we are also hopeful that in the short term, research will provide some ‘quick wins’ in terms of better prevention and earlier opportunities for screening/monitoring/intervention, etc., that can immediately improve the lives of those impacted by the disease. These wins could mean years of additional time for families to be together. Having our own young children and family, prevention and early intervention is a key area for us so we can make sure to do all we can.”

LINDA AND PAUL BARNARD

“My brother, Joe Petitti, had early-onset Alzheimer’s and tragically died at age 53. While early-onset Alzheimer’s only affects a small number of people, we found that the research criteria often excluded those under 60, and thus options for clinical trials were very limited. This motivated Paul and me to connect with an organization dedicated to research in all aspects of the disease. We both have had other family members impacted by both early-onset and late-onset Alzheimer’s.

“There are many ways to support those impacted by Alzheimer’s, but Cure Alzheimer’s stands alone in its commitment to a cure. Without a cure, the need is endless and overwhelming to families and our health care system. It is only through a cure that we can end the suffering that has occurred in our families and so many others. This is where our hope lies.”
PHYL LIS MILLER
Phyllis had experienced firsthand the impact of Alzheimer’s disease on her friends and family. In particular, she witnessed the terrible strain the disease imposed on the families of those suffering from it.

Phyllis first chose to support CureAlz in 2017. It was our unique funding model that resonated with her, and our willingness to embrace multiple research approaches to solving Alzheimer’s disease.

When Phyllis passed in October 2022, we were honored to receive a portion of her estate. We greatly appreciate her bequest and will utilize it to continue to pursue research into a solution for this disease and the devastating impacts that come with it.

MIKE GANG AND THE GANG FAMILY
"My father Stan was a brilliant self-made man. He passed away on March 30, 2016, at the age of 81 from Alzheimer’s disease, and I have thought of him every day since. Dad took a risk and started a business in his early 50s, and he and I grew the business to 750 employees and seven marriages between co-workers—it was a real family. It was the best 14 years of my life, working every day with him!

"Dad was diagnosed with MCI at age 79 and passed quickly, which surprised us, as he was physically very fit, exercised and had a healthy diet. He adored my wife, Erica, who took care of him in his last years, and his four grandchildren. He would light up any time he got to spend time with us. Fortunately, he never forgot the six of us.

"I give because I’m concerned for myself, our family, friends and anyone else who contracts this terrible disease that robs you of life. We need to find a cure, as this devastates families and burdens them financially, emotionally and with the need for constant caregiving time."

THE CROTTY FAMILY FOUNDATION
"Both of my parents had Alzheimer’s," says Shari Crotty. "My mother, Beverley, was diagnosed in 2004 at age 74, although she showed signs of the disease years earlier. She was an amazing woman, kind, sensitive, but unfortunately with Alzheimer’s, she became agitated and fearful. My father, Bob, was diagnosed in 2017 at the age of 88 and died in 2022. He always liked to be in charge and could be very difficult. But with Alzheimer’s he became just the opposite of my mother—funny, sweet and appreciative of those helping him, and he sure did make us laugh until the end.

"In 2006, they were invited to a CureAlz symposium at Massachusetts General Hospital and I attended for them. Rudy Tanzi and Rob Moir were speaking about their research and I left that evening so excited. I knew this organization would do great things for families like mine. I went home and thought I could explain everything I had just heard to Tom—which was more difficult than I thought! When my mother died in 2010, we made our first donation to CureAlz in her memory.

"My husband Tom and I were extremely impressed with the model and knew this was a nonprofit we would like to support on a larger scale. Tom continues to tell other nonprofits about the CureAlz model. We have enjoyed supporting projects for multiple years and seeing our donation make a difference. We are sure the researchers of CureAlz will be involved with finding a cure, and we will stand with CureAlz to help them do so. I hope my children—and all children—won’t need to worry about this terrible disease in the future."
GINA AND TOM VADNAIS

“Tom had a natural talent for business management. He started out at IBM, where he worked for 23 years, and then he was lucky enough to run several other companies. Like many, he was a smart, successful and healthy person. Tennis and golf were lifelong hobbies. Tom had just retired in 2010. By the fall of 2011, our family started noticing thinking issues. A few months later, several signs of short-term memory loss were noticeable. In 2013, we sought medical advice. He died in November 2017.

“Since Tom’s death I am now convinced that it was not Alzheimer’s, but a TBI (traumatic brain injury), which occurred shortly before the problems arose. However, his issues did make me aware of what a devastating disease Alzheimer’s is.

“I learned about CureAlz through a friend. When she told me that 100% would go to research and the Board picks up the [administrative costs], I knew that’s the charity I wanted to support. They have added much knowledge to finding the solutions. There is a long way to go. I continue to give annually, as I believe Cure Alzheimer’s Fund is on the right track.”

KAREN FRIEND

“My connection to Alzheimer’s disease is a very personal one. My late husband Jake, who was diagnosed with early-onset Alzheimer’s in his 40s, died in 2016 at the age of 60. Jake had been an engineer, semi-professional drummer and athlete who was a kind soul loved by many. The disease ravaged his brain and body, but never his spirit.

“I am a researcher in public health who believes the way out of this disease is through research that finds treatments and cures. The more we understand, and the faster empirical findings come to light, the quicker we can end the need for wonderful organizations like Cure Alzheimer’s Fund. I appreciate the dedication and out-of-the-box thinking that has characterized the work of this amazing organization. The findings that have come from Cure Alzheimer’s Fund scientists are nothing short of remarkable. In addition, their consistent, year-over-year 4-star rating in Charity Navigator provides assurance that the money I donate is going directly to supporting the cause to which I am donating.”

LARRY AND JERRY MULLINAX

“Our dad, Ed Mullinax, had Alzheimer’s disease during the last years of his life. Dad was a pioneer in automobile retailing and, in 1975, he invented ‘one-price’ selling. He was an American success story, starting out poor and going on to become the largest Ford retailer in the U.S.A. He was a great leader, a great competitor and a great father.

“After Dad’s passing in 2018, we wanted to help find a cure for this horrible disease that affected our dad and so many others. It was hard to watch what this disease did to our dad, and even harder to watch what Mom had to go through taking care of Dad. Over the last five years we have donated to Cure Alzheimer’s research because we believe it will lead to a cure.”
"My father lived with Alzheimer's disease for 15 years," says Lacy Fyrwald. "It took him away from all of us too early. He was such a fun, smart and important person, and to lose him at such a young age was devastating. There is always a feeling of loss when a loved one is afflicted. But to have Dad physically healthy but unable to engage with us in life—that is a long goodbye. I want to live to see the day when families don't have to mourn the loss of their parents, grandparents, siblings over and over again, while they're alive, because of Alzheimer’s.

"Ernie and I give where we can make the most impact. It's important to us to have a connection to the people who are involved in the organization to which we donate. Cure Alzheimer's Fund has made a huge impact on us because of the people who are dedicated every day to curing this disease. Another important factor to us is how the money is distributed. It's so impressive that the Board covers all the overhead. I love reading about the work CureAlz does and how they are open to ideas. I love everything about this organization."

"Gran suffered from dementia for several years before finally succumbing to the disease. All of the 'hardware' was in good working order, but the 'software' just didn't compute. And God bless our family for being resilient enough to help her and be with her, but that service comes with a large emotional price tag.

"Other organizations provide support and care, and those things are important, but we believe putting an end to Alzheimer’s is the ultimate support. The donation isn’t large, but as we get better at hosting this event and promoting the cause, the donation will get better, too. Plus, it helps to know that 100% of the donation funds research."

Generative art platform Art Blocks hosted reGEN—an exclusive sale of works by five leading artists from around the world supported by Right Click Save. reGEN unites artists and collectors in the fight against neurological disease and, in October, provided the platform for the sale of exclusive works of art to fund research into understanding Alzheimer’s disease. Their auctions resulted in more than $100,000 in contributions to Cure Alzheimer's Fund research. reGEN, curated by Alex Estorick and Foteini Valeonti, included celebrated digital artists Melissa Wiederrecht, Sputniko!, Nat Sarkissian, Robert Hodgin and Marcelo Soria-Rodriguez. Their beautiful and compelling works of art, and the generosity of the artists, are a humbling reminder that Alzheimer's disease does not discriminate. We are grateful for their incredible generosity.
Awareness

Cure Alzheimer’s Fund is committed to increasing knowledge of Alzheimer’s disease by sharing the progress being made through our research grants. In 2023, a new website dedicated to women debuted, events were held that raised awareness, and members of our research community presented recent advances in the prevention, diagnosis and treatment of the disease. We are grateful to everyone who has helped in these efforts.

**WomenandAlzheimers.org Launches**

In honor of National Alzheimer’s Disease Awareness Month in November, Cure Alzheimer’s Fund launched a new website dedicated to the impact of the disease on women. The site provides information on scientific research into understanding sex-based differences and research discoveries through funding by CureAlz. Users can learn about ways to improve brain health—at any age—and read about the remarkable women who are working to find a cure, including researchers, Board members and champions who raise funds for our research.

Visit [WomenandAlzheimers.org](https://www.womenandalzheimers.org) to learn more.
Webinars
Throughout 2023, Cure Alzheimer’s Fund provided the latest updates on progress being made in Alzheimer’s disease research through many informative webinars. Presentations were recorded and can be viewed through the links provided. Visit CureAlz.org to sign up for emails about future webinars.

■ ADVANCING THE FRONTIERS OF ALZHEIMER’S RESEARCH
In this webinar, Dr. Julie Harris, Executive Vice President, Research Management for Cure Alzheimer’s Fund, presented advancements to improve treatment options that are available for people living with Alzheimer’s disease (AD), including an overview of the top factors that contribute to an increased risk of AD, and new diagnostic tools for the disease that show promise. Dr. Harris also reviewed current investigations funded by CureAlz that are exploring immunity and the microbiome.

■ THE CHANGING LANDSCAPE OF ALZHEIMER’S DISEASE: CHALLENGES AND OPPORTUNITIES IN THE ANTI-AMYLOID ERA
A constellation of different pathologies, not just amyloid beta, comes together to damage the brain, leading to cognitive decline and other misfunctions. What are the implications of these pathologies? Dr. Costantino Iadecola of Weill Cornell Medicine gave a presentation that delved into the latest theories around a precision medicine approach to identify major pathologies that cause dementia, and the benefits of using a more holistic approach to brain health: Plan, Protect and Prevent, Preserve.

■ WOMEN AND ALZHEIMER’S DISEASE
Why are women more likely to be diagnosed with Alzheimer’s disease and depression? What is it about our brains that makes women more susceptible to different disorders? Are there some female-specific experiences like pregnancy and menopause that might be driving this increased risk for these disorders? In this conversation with CureAlz CEO Meg Smith, Dr. Liisa Galea of the Centre for Addiction and Mental Health presented her fascinating work in the study of sex-based differences, and her findings on the potential causes of Alzheimer’s disease.

■ ARTIFICIAL INTELLIGENCE IN CLINICAL RESEARCH OF ALZHEIMER’S DISEASE
Technological advancements have resulted in a wealth of available health data. Dr. Ali Ezzati described his work at the University of California, Irvine, using artificial intelligence and machine learning techniques that effectively utilize this abundant data to enhance diagnostics, prognosis, prediction and treatment decisions. His approach to using big data has the potential to benefit all patients by improving health care outcomes.
THE POTENTIAL FOR HARNESING THE MICROBIOME TO PREVENT AND TREAT ALZHEIMER’S DISEASE

In this webinar, Dr. Laura Cox provides insights into the role of the gut microbiome and brain health, including the link between aging, diet, antibiotics, lifestyle and sex-specific interactions that affect the microbiome and influence the immune system. Dr. Cox, a researcher at Brigham and Women’s Hospital and Harvard Medical School, also provides her perspective on future microbiome therapeutic approaches for Alzheimer’s disease.

ALZHEIMER’S DISEASE AND POTENTIAL FUTURE THERAPIES

At the invitation of The Society of the Four Arts in Palm Beach, Florida, Dr. Rudy Tanzi of Massachusetts General Hospital provided a presentation on the latest in Alzheimer’s disease research, diagnostic tools, and new therapeutics and treatments. He spoke about using lifestyle interventions—the S.H.I.E.L.D. plan—to take care of your brain health now and reduce the risk for Alzheimer’s in the future.

The 3rd Annual Cure Alzheimer’s Fund Golf Tournament

On a perfect fall day in October, 104 golfers came together to enjoy a day of golf at the Fishers Island Club in New York and to contribute to research being funded by Cure Alzheimer’s Fund. Fishers Island is among the most exclusive and scenic golf courses in the world and offers picturesque views of Long Island Sound and the Atlantic Ocean from every hole.

Hosted by Henry McCance, Co-Founder and Chair of Cure Alzheimer’s Fund, the full day began with golfers arriving on the island by morning ferry, followed by a hearty breakfast and 18 holes of golf. The tournament was a best ball format, with prizes awarded for top three team scores, longest drive and closest to the pin. Dinner on the lawn rounded out the day before golfers departed the island for a sunset cruise back to the mainland.

Much gratitude to Fishers Island Club for its generosity in providing this unique opportunity to raise funds for research.
There are many ways to be part of the solution to find a cure. It is through the support of our donors that we move a little closer every year to understanding Alzheimer’s disease and finding a cure.

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 310
Wellesley Hills, MA 02481

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 #52-2396429.

Online
Make a secure gift online by credit card, PayPal, Venmo or cryptocurrency.
bit.ly/CureAlzDonate

Mail
Mail your check payable to Cure Alzheimer’s Fund and send to Cure Alzheimer’s Fund, 34 Washington St., Suite 310, Wellesley Hills, MA 02481

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

Give Monthly
Monthly giving is a powerful and enduring way to support research to cure Alzheimer’s disease. To select a specific gift amount for automatic, recurring contributions, visit bit.ly/CureAlzDonate.

Qualified Charitable Distribution (QCD)
A QCD paid from your Individual Retirement Account (IRA) allows you, if you’re 70½ or older, to donate up to $105,000 to Cure Alzheimer’s Fund directly from your IRA. And it’s 100% tax free. bit.ly/3PN49qQ

Securities and Wire Transfer
Securities such as stocks, mutual fund shares or other appreciated assets can be a tax-efficient way to make a gift to Cure Alzheimer’s Fund. Gifts may be sent electronically via wire transfer. Contact us at Info@CureAlz.org for instructions.
Donor Advised Funds (DAF)
A DAF is a charitable savings account that gives you flexibility to recommend donations to Cure Alzheimer’s Fund. With funds held by Fidelity Charitable, Charles Schwab or Great Kansas Community Foundation, use the DAF form on our website. bit.ly/DAFform

For all other DAF holders, please mail checks to: Cure Alzheimer’s Fund, 34 Washington St., Suite 310, Wellesley Hills, MA 02481

Be a Fundraiser
If you would like to host your own event or fundraiser, email us at Hero@CureAlz.org to learn more and how to get started.

Secure Your Legacy
The Legacy Society honors those who make a gift to Cure Alzheimer’s Fund through their will or estate plans. In 2023, 25% of the research grants provided to scientists throughout the world were enabled by generous donors who included CureAlz in their estate plans. Please consider joining our Legacy Society; your contribution will ensure your generosity beyond your lifetime.

To learn more about how to include Cure Alzheimer’s Fund in your will, please contact us at 781-237-3800 or Legacy@CureAlz.org.

DONATE TODAY
To explore these and other ways to give, scan the QR code, visit bit.ly/GiveCureAlz, email us at Info@CureAlz.org or call 781-237-3800.
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