

FUNDING WORLD'S MPORTANT RESEARCH



MISSION

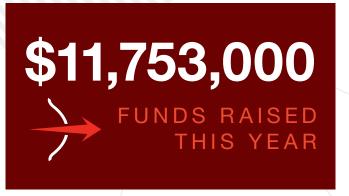
To fund research with the highest probability of preventing, slowing or reversing Alzheimer's disease.

2015 ANNUAL REPORT

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PISCAL YEAR 2015

BY THE NUMBERS





9,128
DONORS



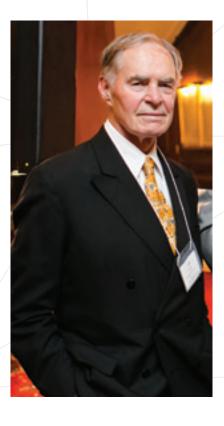












Message from the Chairman

Jeff Morby, Chairman and Co-Founder
Cure Alzheimer's Fund

Dear Friends,

At a recent meeting of our Research Consortium, made up of some of the world's most brilliant scientists in the field of Alzheimer's research, there was virtually unanimous agreement among the participants that CAF has been one of the principal leaders in the field and is unique among the research groups grappling with the disease.

The above described perception has motivated me to share with you what I believe are five important leadership roles CAF has carried out within the field:

First: Leadership in the Genetics of Alzheimer's Disease

Twelve years ago, when we first established the foundation, there were only 4 known Alzheimer's genes, representing about 30% of the causes of the problem. We knew that we had to find and understand the many other undiscovered genes in order to make headway against the disease. Consequently, in 2006 we performed the first genomic scan of the disease and made a truly breakthrough discovery at the time—we discovered 5 new genes, a discovery which was dubbed one of the "Top 10 medical breakthroughs in 2008" by *TIME Magazine*. Subsequently we were the first in 2011 to perform another genomic scan using the newest technology at the time. After that, in 2013, we were the first organization in the world to use new state-of-the-art technology "whole genome sequencing" to sequence the entire DNA of a database of more than 1,500 individuals from families afflicted by Alzheimer's disease.

Today we have one of the largest databases in the world of the genomics of Alzheimer's disease, having discovered more than 50 new Alzheimer's genes containing over 350 genetic variants associated with risk for the disease. As Rudy Tanzi will describe, this database has allowed us to undertake the **Genes to TherapiesTM (G2T) program,** in which we have launched a major effort to understand 20 of the most significant Alzheimer's genes—how they interact individually and collectively to cause AD—and how we can stop them from doing so.

Second: Leadership in the Creation of Advanced Research Tools

Of course, the genomic database combined with the genomic analytics is our major research tool. But, also importantly, Rudy, Doo Yeon Kim, and their team have developed a breakthrough technology, called "Alzheimer's in a Dish" (ADD), which allows scientists for the first time to grow a neuron in a 3-dimensional environment, a mini-brain, in order to see how the neuron develops its pathology and how it reacts to potential medications. The analytical tool is so powerful that it can be used for analysis of a variety of other neurological diseases. Its importance was underlined both by a full-page article in *The New York Times* as well as a special blog post by Francis Collins, director of the NIH.

ADD is also leading CAF to develop additional tools. One of these represents an extension of the mini-brain concept to include visualization of the formation and spreading of Tau, the actions of microglia, and the operation of the bloodbrain barrier.

Another is the creation of rapid throughput analysis processes, using ADD, to ascertain whether or not existing drugs currently on the market can be repurposed to be used as preventatives for Alzheimer's disease. We have already analyzed approximately 1,200 drugs and have identified dozens of high-potential prospects for consideration in future trials.

Finally, an important analytical tool for most of biological science is what is called a "transgenic mouse," which is a mouse grown with human DNA, which causes such mice to neurologically respond to stimuli as a human brain would do with the same set of genes. Such mice, with Alzheimer's DNA, can be used to test potential drugs for use against Alzheimer's disease. As mentioned earlier, in 2004, there were only 4 known AD genes. These same genes, incorporated into the brains of transgenic mice, have been for more than 10 years the "mice standard" against which potential drugs have been tested. However, we now know that there are far more AD genes than 4. Therefore, many more transgenic mice beyond those of the 4 genes are required if we are to have the proper vehicles for testing drugs. So, as part of the G2T project, we are now developing new transgenic mice with the information provided by our genomic database. Importantly, as we develop such mice, we make them available to both our researchers and the whole scientific community - a major contribution to science.

Third: Leadership in the Development of Effective Forms of Scientific Collaboration

It is hard to imagine a more talented group of scientists than the list of our Research Consortium and SAB collaborators. This last year we have added another group of exceptional scientists, as Rudy will describe below. But apart from capabilities, what truly distinguishes these groupings of scientists is their willingness, in fact eagerness, to collaborate. Over time, all of them have developed with us an atmosphere of trust and cooperation and are willing to share their unpublished insights and participate in quarterly brainstorming sessions focused on attacking the disease in new and creative ways. Additionally, they are guided in their research decisions by **CAF's Roadmap**, a jointly shared strategy, which is changed as new scientific insights are attained.

Fourth: Conceptual Leadership in the Attainment of New Scientific Insights

I believe that it is safe to say that we have been a conceptual leader in the field of Alzheimer's research. Our emphasis on genomics, the development of new tools, and our collaborative approach to scientific exploration, as described above, are some examples. But there are others. One of these is the conceptualization of a comprehensive model of Alzheimer's disease. This model has allowed us to identify what we call "intervention points" to use in potentially combatting the disease as it spreads. Each one of these intervention points represents a stage of development of the disease, which stage, if stopped, will arrest the progression of the disease. We have organized our research around those intervention points and the G2T project, and this year have committed to fund 44 different research projects totaling \$10 million.

Of significant importance to the understanding of Alzheimer's disease is our new conceptualization of what is called the "Anti-Microbial Protection Hypothesis." Rob Moir and Rudy Tanzi of Mass General have been working for 5 years to validate this concept, and their paper providing strong evidence for the hypothesis in various experimental models was published in *Science Translational Medicine* in May 2016. This revolutionary hypothesis has the potential to fundamentally alter the current paradigm regarding how Alzheimer's pathology is triggered in the brain. They have shown that Abeta is a component of the brain's innate immune system, which is protective of the brain. Abeta traps pathogens in plagues when they enter the brain and kills

Twelve years ago, when we first established the foundation, there were only 4 known Alzheimer's genes, representing about 30% of the causes of the problem... Today we have one of the largest databases in the world of the genomics of Alzheimer's disease, having discovered more than 50 new Alzheimer's genes containing over 350 genetic variants associated with risk for the disease.

them, which is a good thing and essential for the protection of the brain. However, too much of this activity, or genetic defects, may cause the brain to overproduce Abeta or fail to clear Abeta, thereby leaving too much Abeta in the brain. This results in "tangles" which kill neuronal cells from within, ultimately leading to Alzheimer's disease. This radical, new view has major implications for drug discovery.

As new discoveries are made in the field, we adjust our priorities. One example of that is the human "microbiome" and what is called "epigenetics." Based on solid scientific evidence, we now know that our human microbiome (made up of bacteria and other microbes in our gut and elsewhere within our body) has the capacity to influence how our genes code for proteins, with major implications for the disease. The gut microbiome also regulates brain inflammation, a key pathological feature of Alzheimer's disease. We have already undertaken two new research projects with leaders in that field.

Fifth: Leadership in Foundation Management (Venture Philanthropy)

Many of the founders of this organization are former venture capitalists. The founders and directors have, over time, personally contributed \$23.8 million to the foundation. These monies are used to pay the operating expenses of the foundation so that any contributions received from third parties go 100% into research. Given our backgrounds and financial contributions to the organization, we don't want to waste money or waste time. We want a cure or preventative as soon as possible, and we manage the institution with this aim. We call the way we manage "Venture Philanthropy." And the management processes we have set up have distinguished us as risk takers, fast decision makers, and strategists. These processes represent one of the reasons for our success (and one of the reasons we have been given a four star rating by Charity Navigator for the fifth consecutive time).

Cures in the Pipeline

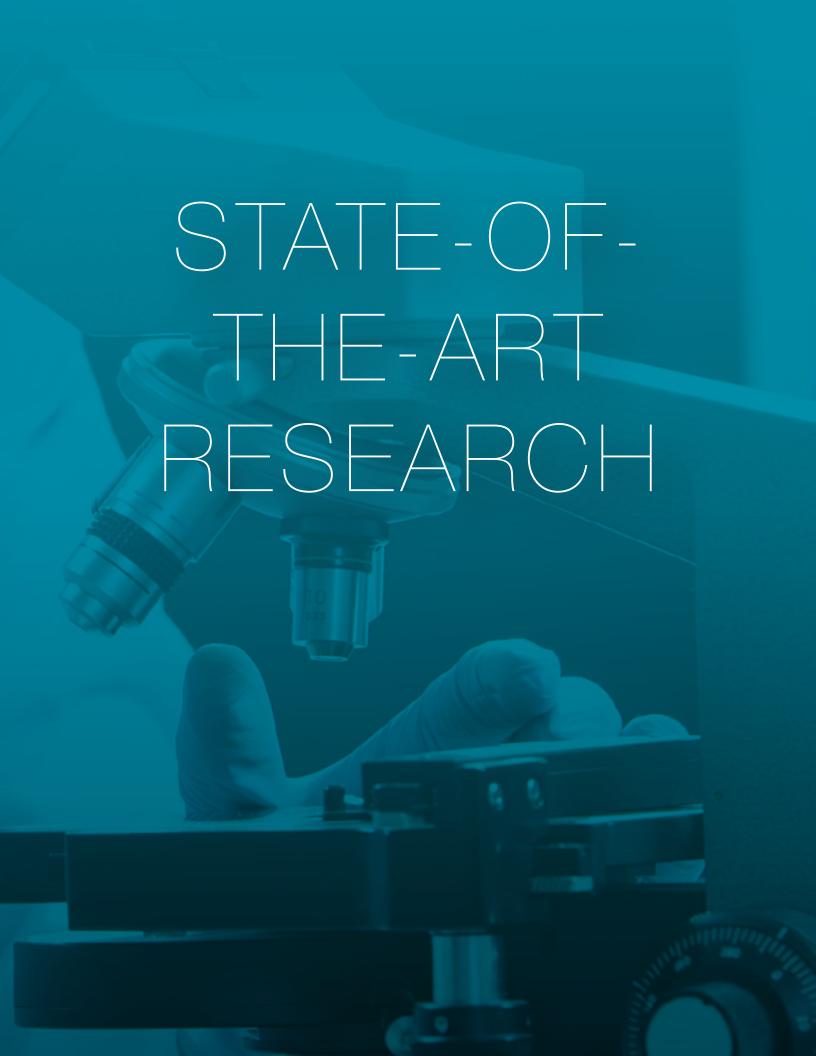
Potential cures/preventatives have been long in coming, but we now have two in the pipeline. One of these, a gamma secretase modulator, is the equivalent of a statin for AD. It is, in essence, a preventative for Abeta accumulation in the brain in much the same way a statin is a preventative for the over-accumulation of cholesterol in the arteries. The second, Amylyx, is a medicine developed to protect neurons from cell damage which might occur as a result of overconcentration of Amyloid in the brain and brain inflammation. We expect Amylyx to go to human trials in 2016 and the gamma secretase modulator in early 2017. We also have preliminarily identified numerous existing medications which could possibly be repurposed for Alzheimer's disease and, of course, have underway a great number of scientific studies, any one of which could identify a promising new drug at any time.

Many, Many Thanks

All of the above would not have been possible without your support and the support of the more than 23,000 contributors to our organization. In 2015 we raised \$11.7 million, our 11th record fundraising year. Thanks to all of you very much for your significant generosity. A thank you, also, to our wonderful researchers and staff, all of whom continue to be inspired by our quest to rid this planet of the dread disease and as a result produce inspirational results, which continue to amaze.

Very Best Wishes,

Jeffrey L. Morby Chairman and Co-Founder





Message from the Research **Consortium Chairman**

Rudolph E. Tanzi, Ph.D., Chairman, Research Consortium, Cure Alzheimer's Fund

Dear Cure Alzheimer's Fund Friends,

2015 was another great year for the research funded by Cure Alzheimer's Fund (CAF). I am very pleased to report that we capitalized on our previous momentum and made immense progress.

During the year, we added many new esteemed colleagues to our Research Consortium and our Scientific Advisory Board, as well as collaborators and grantees. These additions to our already significant group of accomplished scientists has the impact of substantially expanding our research efforts while maintaining our standards for only funding research of the highest quality and with the greatest potential impact on understanding and treating Alzheimer's disease (AD).

For 2015, we increased our funded research to over \$10 million, double the amount from 2014 of just over \$5 million.

I am confident in stating that Cure Alzheimer's Fund is operating at an entirely new level of research excellence, which is unparalleled in the field of Alzheimer's disease research.

Most of our projects remain focused on the Genes to Therapies™ (G2T) initiative, which is now in high gear. G2T involves taking the top Alzheimer's disease genes, including the previously established (4) as well as a dozen of our new ones, and using them to create new disease models to study AD. The primary purpose of G2T is to translate our unprecedented database of novel genetic results into a deeper understanding of the causes of Alzheimer's disease as well as novel drug discovery and development for treating and preventing the disease.

In addition to studying the above Alzheimer's disease genes in various mouse and cell models, we are incorporating the new disease gene-derived data into our 3D stem cell-derived neuronal cell cultures (Alzheimer's in a Dish). We have also used Alzheimer's in a Dish to initiate the 3D Drug Screening (3DDS) project with several collaborators. Our approach is to use 3DDS to screen all existing approved drugs (~1200) and many clinically safe, but not yet approved, investigational drugs. The objective is to evaluate which drugs can be repurposed to stop beta-amyloid deposition, tangle formation and neuronal cell death in 3D cultures. The studies have already resulted in the identification of several drugs that appear to block tangle formation. In other studies, we are screening for drugs that will stop neuroinflammation using the AD genes involved in innate immunity in the brain, such as CD33. And, we are developing novel 3D systems to address innate immunity and the blood-brain barrier (BBB) in Alzheimer's disease.

In addition to screening for new drugs, there are other projects in development that will take drugs funded by Cure Alzheimer's Fund into clinical trials in patients with Alzheimer's disease. Most notably, these include the gamma secretase modulators (GSM), aimed at lowering Abeta levels, and the Amylyx compounds, aimed at protecting neurons from neuroinflammation. This research effort is currently being conducted and will continue through 2016.

The Cure Alzheimer's Fund portfolio of projects aimed at diagnosis and detection of Alzheimer's disease has been steadily increasing, especially pre-symptomatically for the purposes of early prediction, early detection and early intervention.

An area of great growth is in the study of the role of neuroinflammation and innate immunity in the pathological pathways of Alzheimer's disease. In addition to the G2T projects focusing on several new AD genes involved in these pathways, such as CD33 and TREM2, we are also investigating how microglial cells and astrocytes contribute to the death of nerve cells in AD. Cure Alzheimer's Fund was one of the first foundations to appreciate the key role played by innate immunity in Alzheimer's disease, when we discovered in 2008

that CD33 is an AD gene. Today, entire AD research programs around the world are focused on CD33 and its counterpart gene, TREM2, first discovered by the recipient of a grant from Cure Alzheimer's Fund.

As an extension of studies of innate immunity in the brain and its role in AD, our research has been advancing our ongoing studies of the role of microbial organisms, such as bacteria, viruses and fungus (yeast) in Alzheimer's disease pathology. The ongoing work has resulted in a very exciting new paper, now in press. This paper is poised to rock the very foundation of our current models of the etiology and pathogenesis of Alzheimer's disease. We have now shown in several different animal models and cell culture models (mice, fruit flies, dirt worms and neurons) that beta-amyloid is clearly an anti-microbial substance produced in the brain to protect against infection, including yeast (candida), herpes simplex virus 1 and various bacteria.

Our research has demonstrated that by infecting the brain of a very young (one-month-old) AD mouse model, in which no plague would normally be present until 6-8 months of age, abundant amyloid deposition could be seeded in the brain, virtually overnight. Moreover, each amyloid plague that formed overnight was observed to contain at its center a single bacterium. The resulting theory is that, in the brain, just a single bacterium that gains entry across the blood-brain barrier can lead to a senile plaque. As revealed with Alzheimer's in a Dish, amyloid deposition can then trigger tangles to form in neighboring nerve cells, leading to cell death. In view of these results, we are now actively investigating our hypothesis that as we age, low-grade and clinically non-symptomatic infections of the brain from viruses, bacteria and yeast, trigger beta-amyloid deposition in the brain as a protective mechanism. Beta-amyloid then leads to tangle formation, followed by cell death, inflammation and, ultimately, dementia. If this hypothesis is borne out, we can envision potentially stopping the pathological process of Alzheimer's disease at its earliest pre-symptomatic stage, by therapeutically targeting specific microbial infections in the brain.

In summary, this has been a landmark year for Cure Alzheimer's Fund. Not only are we supporting some of the most exciting and state-of-the-art research in the field of Alzheimer's disease, we have also dramatically expanded our team of investigators. We now have the breadth, depth and focus in our research portfolio to move forward with true momentum.

The other role of microbes in Alzheimer's disease involves beneficial microbes that make up our "microbiome." The microbiome is the total collection of the several thousand species of bacteria that live in our gut, on our skin and in body cavities. In particular, the gut microbiome is directly connected to the brain and has been shown to affect mood and neuroinflammation in the brain. Two projects have now been funded by Cure Alzheimer's Fund to explore the role of the gut microbiome in Alzheimer's disease. The goal of these studies is to determine how we might treat and prevent AD, and otherwise enhance brain health, by managing the gut microbiome.

As 2015 came to a close, we had a new paper accepted describing (3) new Alzheimer's disease genes, all of which offer new targets for drug discovery, including one involved in cholesterol metabolism and two others that appear to be involved in tangle formation. We have also finalized our whole genome sequencing data to arrive at roughly 350 different gene mutations and variants in about 50 genes that directly affect risk and/or age-at-onset for Alzheimer's disease. Our growing database of detailed genomic data on AD continues to be the most comprehensive and highest quality, worldwide. We are currently preparing the publication of our unprecedented whole genome sequencing data and plan to make all of it available to the entire research community.

In summary, this has been a landmark year for Cure Alzheimer's Fund. Not only are we supporting some of the most exciting and state-of-the-art research in the field of Alzheimer's disease, we have also dramatically expanded our team of investigators. We now have the breadth, depth and focus in our research portfolio to move forward with true momentum.

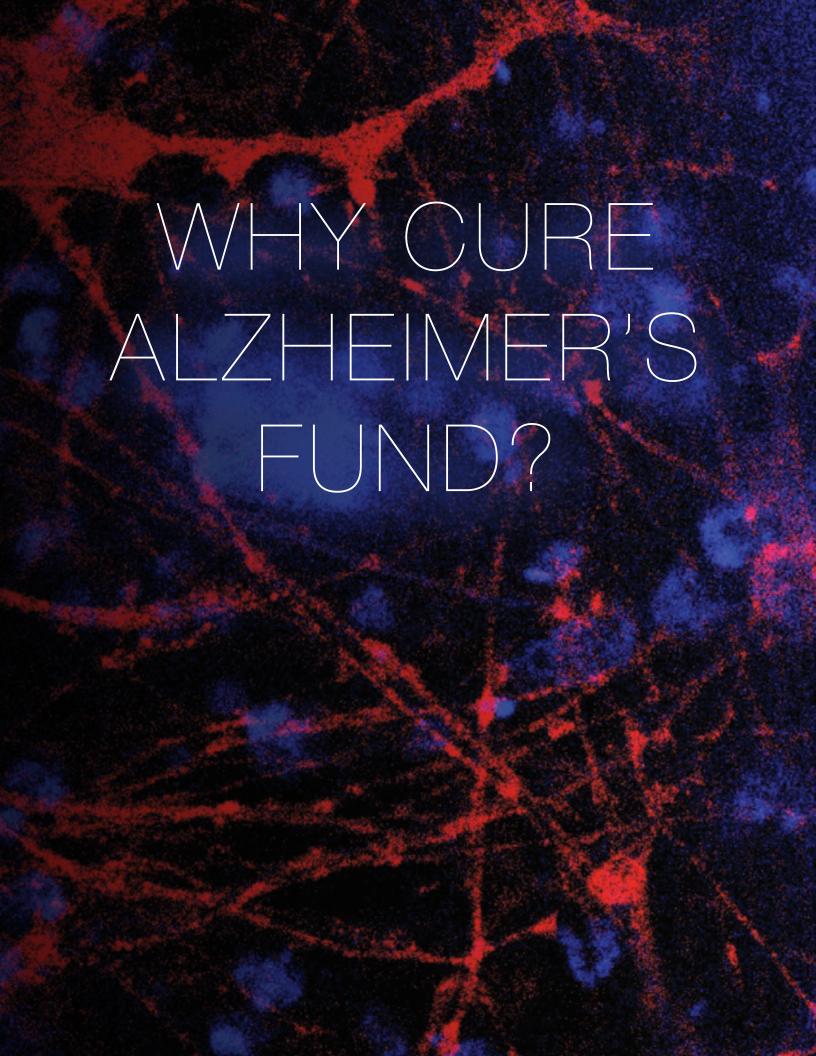
2015 was a very exciting year, and 2016 is even more so. Our work is only possible because of the generosity of all of those who have donated to and supported Cure Alzheimer's Fund. Thank you to my colleagues, to the Board and staff of Cure Alzheimer's Fund. We are all enthusiastic about our progress and know that our work continues. We will not stop until we have discovered a way to end Alzheimer's disease.

Thank you,

Rudy

Rudolph E. Tanzi, Ph.D. Joseph P. And Rose F. Kennedy Professor of Neurology Harvard Medical School

Vice Chair, Neurology Director, Genetics and Aging Research Unit Massachusetts General Hospital



ALZHEIMER'S: A DEVASTATING DISEASE THAT MUST BE STOPPED

Alzheimer's disease is the most common cause of dementia worldwide, affecting more than five million individuals in the United States alone. It is a progressive disease of the brain that develops slowly over time and leads to increasingly serious cognitive decline, causing problems with learning and memory, inducing personality changes and impairing motor skills. The disease is associated with the presence of amyloid plagues, tau tangles and inflammation in the brain, and it is always fatal. Alzheimer's primarily affects the elderly, but it can also emerge as an early-onset form in middle age. With more than 70 million aging baby boomers, Alzheimer's disease has the capacity to engulf and even bankrupt national healthcare systems in the coming decades.

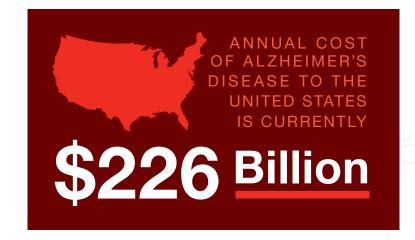
Even with adequate funding, finding medical solutions for this disease will not be easy. Alzheimer's pathology starts 20 years or more before symptoms become noticeable, and scientists now believe that we need to tackle Alzheimer's in these presymptomatic stages in order to fight the disease effectively.



PICTURED LEFT: Neurons

Abeta plaques

By redirecting skin cells from Alzheimer's patients and turning them into nerve cells, we are able to study adult Alzheimer's neurons in the lab.



CURE ALZHEIMER'S FUND FINDS INNOVATIVE WAYS TO MOVE RESEARCH FORWARD

Cure Alzheimer's Fund (CAF) finances high-potential research, some of it in the "proof of concept" stage, which might not be funded initially by the National Institutes of Health (NIH) or other organizations. We support the scientists who are doing the most innovative work on Alzheimer's pathology-highpotential research that also carries some risk. CAF's "pump priming" approach is proving increasingly successful, as more of our early-stage grants are leveraged into substantial, longer-term funding by NIH and others.

Cure Alzheimer's Fund was created in late 2004 with a mission to end the disease by:

- 1. Identifying all risk genes;
- 2. Investigating those genes to reveal underlying disease mechanisms: and
- 3. Aggressively pursuing potential therapies based on the knowledge gained from studying Alzheimer's genes.

Using our genetic discoveries as guideposts, Cure Alzheimer's Fund has sponsored dozens of studies investigating the central mechanisms of action behind the disease. Fully 100 percent of funds raised by Cure Alzheimer's Fund go directly to research—the Founders and Board of Directors covers all overhead expenses. Our unique funding strategy is focused on preventing, slowing or reversing the disease. Our ultimate objective is to stop the disease.

100% of **ALL** donations **DIRECTLY FUND RESEARCH**





Since our founding, Cure Alzheimer's Fund has contributed more than \$38 million to research, and CAF-funded initiatives have been responsible for several key breakthroughs. We support many of the best scientific minds in the field of Alzheimer's research, and we do it without any financial gain for our founders or donors.

Our Research Consortium is an all-star team of scientists working at premier research institutions around the globe, regularly conferring with one another on the progress and challenges in their research and to share their findings even before publication.

Cure Alzheimer's Fund has dedicated substantial resources to identifying the full complement of Alzheimer's genes. The Alzheimer's Genome Project™ was launched in 2005—and the study's first phase led to the identification of more than 100 new Alzheimer's candidate genes. Our Genes to Therapies™ (G2T) initiative will determine how the top-priority genes affect Alzheimer's pathology, thus leading to faster development of effective interventions. These initiatives, combined with many more promising research projects, will help bring us closer to developing effective therapies and, eventually, finding a way to prevent and cure the disease.

Please visit our website at curealz.org to join us in the quest for a cure.

PICTURED ABOVE:

- Neurons with neurofibrillary tangle-like structures
- Green Fluorescent Protein (GFP)

An important outcome of Cure Alzheimer's Fund's stem cell research was the development of "Alzheimer's in a Dish," which confirmed the long-debated amyloid hypothesis by allowing researchers to see precisely how amyloid stimulates the creation of tau tangles.

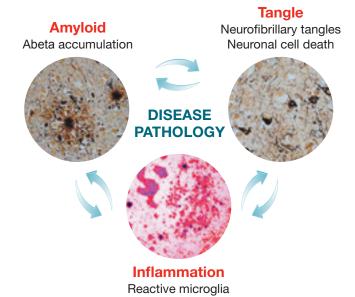
OUR RESEARCH IS

Cure Alzheimer's Fund has a focused plan to end Alzheimer's disease, and we make significant strides towards that goal each year. Our research has contributed to a more thorough understanding of how Alzheimer's pathology progresses from the earliest to latest stages of the disease, including the identification of key genes and the functions of these genes.

Alzheimer's Disease Model

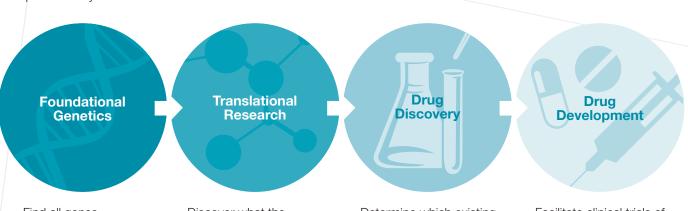
This model of Alzheimer's disease allows us to identify three basic strategies for intervention in the process:

- 1. An early-stage intervention inhibiting the production of the Abeta protein (the primary component of plagues characteristic of Alzheimer's disease) and/or clearing it from the brain after it forms.
- 2. An early- to mid-stage intervention that would inhibit the formation of tau tangles (proteins abundant in the central nervous system that have become defective and twisted into microscopic strands) and protect neurons from undue stress.
- 3. A late-stage intervention that would fight inflammation and thus slow down or even stop the disease process.



Research Roadmap

By addressing Alzheimer's at its origins and finding the major causes of the disease, we are accelerating developments that may lead to effective therapies. The projects we fund are based on this research roadmap, which we believe to be the quickest way to a cure.



Find all genes that contribute to risk factors for or protection against Alzheimer's disease; prioritize those with the greatest impact. Discover what the previously known Alzheimer's genes can teach us about Alzheimer's disease pathology and determine the role of the newly identified genes.

Determine which existing drugs or novel chemical compounds most safely and effectively disrupt the Alzheimer's pathology generated by the highest priority genes.

Facilitate clinical trials of the most effective drugs by partnering with biotech firms or pharmaceutical companies to hasten drug development and approval.

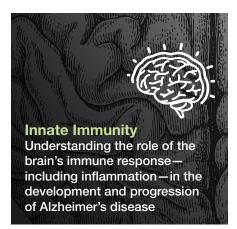
MAKING A DIFFERENCE

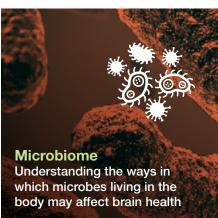
9 Areas of Research Focus

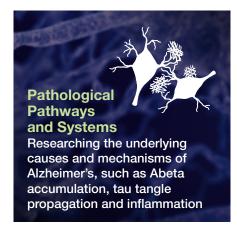
The four roadmap categories focus our funding and have helped us make significant progress in our understanding of the disease. This growing understanding has allowed us to further delineate our roadmap into the following areas of focus:





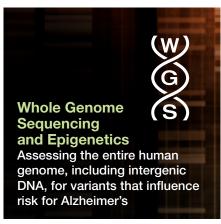














By funding the most promising research in each of these categories, we will build an in-depth and multifaceted picture of Alzheimer's disease. Approaching the disease from these perspectives will allow convergence on the ultimate goal—a cure.

Go to **curealz.org/focus-areas** to learn more about where we are focusing our research.

YOUR SUPPORT HELPED US MAKE A

CURE ALZHEIMER'S FUND DEDICATED MORE THAN \$10 MILLION TO RESEARCH FUNDING

Cure Alzheimer's Fund provides grants to support high-potential research that continues to gain momentum and break new ground in the field. And we know that our model is working.

2015 was a significant milestone year for our organization. We distributed more than \$10 million to 44 of the most innovative and exciting Alzheimer's research projects in the world (see pages 18-19 for a complete list of our 2015 research projects). This \$10 million—a direct result of your support and dedication to helping us prevent, slow or reverse this fatal disease—is nearly double what we were able to distribute to research in 2014.

Rudy Tanzi and Doo Yeon Kim's Alzheimer's in a Dish study, which replicates human brain cells in a petri dish for use in Alzheimer's research, is one of the biggest breakthroughs recently funded by Cure Alzheimer's Fund. The project has gained recognition by the National Institutes of Health (NIH), the medical and research communities, news media and others for this ground-breaking discovery that continues to propel forward our understanding of the disease. In 2015, Smithsonian Magazine recognized Tanzi and Kim by bestowing its prestigious American Ingenuity Award upon the scientists for their work on this project and their revolutionary breakthroughs in science.

Cure Alzheimer's Fund provided grants to a landmark number of other innovative, high-potential research projects throughout 2015, spanning almost all of our nine focus areas. From Alexandra Newton's research on cancer and Alzheimer's to Berislav Zlokovic's new insights into the blood-brain barrier to Rob Moir's work on the innate immune system—and more—CAF-funded research made great strides and tremendous impact in the Alzheimer's field this year.

More than 200 people gathered in Boston at our fifth annual Research Symposium in mid-October, and another 500 tuned in online, to hear about the impact and excitement that our research projects are generating in the field and beyond. The event highlighted our growing understanding of the Alzheimer's disease process from our nine areas of focus as well as the research, collaboration and breakthroughs of the past year.

Congress and the President continue to support the need for legislation to improve the process of finding cures for life-threatening diseases like Alzheimer's. In July, the House of Representatives passed the 21st Century Cures Act. which calls for increased investment in NIH with a focus on innovation, speed, safety and commitment to improving the lives of people suffering from deadly diseases. Cure Alzheimer's Fund worked closely with both Republicans and Democrats throughout the legislative process to improve the bill. And at the end of the year, Congress and the president approved an additional \$350 million in NIH funding for Alzheimer's disease research in FY16. We still have a long way to go to get to the \$2 billion a year in research funding necessary to meet the goals outlined in the National Plan to Address Alzheimer's Disease, but this commitment by Congress and President Obama is a significant step toward reaching that goal.

MAJOR IMPACT IN 2015



scientists doing the most innovative work on Alzheimer's pathology-high-potential research that also carries some risk, as it still might be in the "proof of concept" stage. This "pump priming" is proving increasingly successful, as more of our grants are leveraged into more substantial and longer-term funding by the National Institutes of Health (NIH) and others.

Since we began in 2004, our investment of more than \$38,000,000 has resulted in more than \$59,000,000 in NIH and other foundation grants, for a total of more than \$97,000,000 going to Alzheimer's disease research.

2015 federal grant recipients include:

- Gal Bitan, Ph.D.
- Guojun Bu, Ph.D.
- Marco Colonna, M.D.
- Caleb Finch, Ph.D.
- Charles Glabe, Ph.D.
- Ana Griciuc, Ph.D.
- David Holtzman, M.D.
- Robert Malenka, M.D., Ph.D.
- Rudy Tanzi, Ph.D.
- Giuseppina Tesco, M.D., Ph.D.
- Gopal Thinakaran, Ph.D.
- Robert Vassar, Ph.D.
- Steven Wagner, Ph.D.
- Benjamin Wolozin, M.D., Ph.D.
- Zhongcong Xie, M.D., Ph.D.
- Berislav Zlokovic, M.D., Ph.D.

OUR RESEARCHERS HAVE PUBLISHED 190 PAPERS, WHICH HAVE BEEN CITED OVER

10,300

For a full listing of published papers supported by Cure Alzheimer's Fund, see curealz.org/ research/published-papers.

We thank all of the researchers and lab personnel for their dedication and commitment to the common mission of ending this disease, and we congratulate them on the important insights their published work reflects.

We are inspired by the progress and milestones achieved in 2015 and remain dedicated to making possible the most impactful research advancements to stop Alzheimer's disease before it even starts.

RESEARCH PROJECTS

Cure Alzheimer's Fund distributed \$10 million to support 44 research projects across our focus areas, an all-time high that allowed us to fund even more of the most innovative research in 2015.

Visit curealz.org/research/ funded-research to read the complete research abstracts for these projects.

Project/Researcher **Distribution Amount**

Froject/ nesearcher	DISTRIBUTION ATTIOUR
Genes to Therapies/Drug Screening	
ABCA7 in Brain Homeostasis and Alzheimer's Disease Guojun Bu, Ph.D., and Takahisa Kanekiyo, M.D., Ph.D., Mayo Clinic Jacksonville	\$200,000
3DDS: A 3-D Human Neural Cell Culture System for Studying Neuron-Microglia Interaction in Alzheimer's Disease Hansang Cho, Ph.D., University of North Carolina at Charlotte	\$150,000
Role of Blood-Brain Barrier Function in Alzheimer's Disease Pathogenesis Investigated Using a 3-D Microfluidic Platform Se Hoon Choi, Ph.D., Massachusetts General Hospital, and Roger D. Kamm, Ph.D., Massachusetts Institute of Technology	1 \$291,056
Discovery of CK1 Activators for Inducing the Autophagic Degradation of APP Beta-CTF Paul Greengard, Ph.D., The Rockefeller University	\$450,000
Investigation of sTREM2 in CSF as a Potential Biomarker for Neuronal Cell Death Christian Haass, Ph.D., DZNE, Ludwig Maximilians University of Munich	\$144,182
The Biological Impact of TREM Locus Mutations in Alzheimer's Disease David Holtzman, M.D., and Marco Colonna, M.D., Washington University, St. Louis	\$250,000
The Role of the KIBRA Gene in Abeta Regulation of AMPAR Trafficking Richard L. Huganir, Ph.D., Johns Hopkins University	\$100,000
3DDS: 3-D Neural Core/High-throughput Drug Screening for Alzheimer's Disease Using 3-D Human Neural Culture System Doo Yeon Kim, Ph.D., Massachusetts General Hospital, Harvard University	ems \$400,000
The Putative Role of Red Blood Cell CR1 Levels in Abeta Clearance and Alzheimer's Disease Pathogenesis Cynthia A. Lemere, Ph.D., Brigham and Women's Hospital, Harvard University	\$150,000
Extracellular Vesicle-Based Targeting of CD33-Mediated Pathology for Alzheimer's Disease Therapy Casey Maguire, Ph.D., Massachusetts General Hospital, Harvard University	\$150,000
PKC Mutations and Alzheimer's Disease Alexandra Newton, Ph.D., University of California, San Diego	\$220,000
G2T Research Models and Materials Taconic Biosciences Inc.	\$305,069
3DDS: Microglial Core/CD33 and Alzheimer's Disease: From Biology to Therapy Rudy Tanzi, Ph.D., and Ana Griciuc, Ph.D., Massachusetts General Hospital, Harvard University	\$400,000
Alzheimer's Genome Project Rudy Tanzi, Ph.D., Massachusetts General Hospital, Harvard University	\$1,500,000
Functional Characterization of GGA3 Mutations Associated with Alzheimer's Disease Giuseppina Tesco, M.D., Ph.D., Tufts University	\$150,000
BIN1 in Alzheimer's Disease Neuropathology Gopal Thinakaran, Ph.D., University of Chicago	\$150,000
Studying the Functional Consequences of Alzheimer's Disease Risk Variants in the CLU and ABCA7 Genes Using Both Human and Mouse Models Li-Huei Tsai, Ph.D., Massachusetts Institute of Technology	\$250,000
Molecular and Cellular Mechanisms of ACE1 Variant in Alzheimer's Disease Robert Vassar, Ph.D., Northwestern University	\$250,000
G2T: Centralized Research Core Operations Management Wilma Wasco, Ph.D., Massachusetts General Hospital, Harvard University	\$185,350
3DDS: High-Content Drug Screen Using a Novel 3-D Cell Model of Alzheimer's Disease Stephen Wong, Ph.D., Houston Methodist Research Institute	\$150,000
3DDS: Alzheimer's Disease Drug Discovery in 3-D Weiming Xia, Ph.D., Boston University	\$150,000
PICALM Gene Therapy and Drug Screening for Abeta Clearance Berislav Zlokovic, M.D., Ph.D., University of Southern California, and Beverly L. Davidson, Ph.D., University of Pennsylvania	\$375,170
The Role of PICALM Mutations in Alzheimer's Disease Berislav Zlokovic, M.D., Ph.D., and Zhen Zhao, Ph.D., University of Southern California	\$100,000

Project/Researcher	Distribution Amount
Identification and Early Detection	
Discovery of Alzheimer's Disease Blood Biomarkers Using Phage Display Technology, Year 2 Yueming Li, Ph.D., Memorial Sloan Kettering Institute	\$100,000
Innate Immunity	
Abeta Expression Protects the Brain from Herpes Simplex Virus Robert D. Moir, Ph.D., Massachusetts General Hospital	\$325,000
Targeting Beneficial Innate Immunity in Alzheimer's by IRAK-M Deletion Terrence Town, Ph.D., University of Southern California	\$150,000
Microbiome	
Role of the Gut Microbiome in Alzheimer's Disease Pathology and the Potential of Probiotic Therapeutic Strategies Deepak Kumar, Ph.D., Massachusetts General Hospital, and Robert D. Moir, Ph.D., Massachusetts General Hospital, Harvard University Company of the Gut Microbiome in Alzheimer's Disease Pathology and the Potential of Probiotic Therapeutic Strategies Deepak Kumar, Ph.D., Massachusetts General Hospital, Harvard University Company of the Gut Microbiome in Alzheimer's Disease Pathology and the Potential of Probiotic Therapeutic Strategies Deepak Kumar, Ph.D., Massachusetts General Hospital, and Robert D. Moir, Ph.D., Massachusetts General Hospital, and Potential of Probiotic Therapeutic Strategies Deepak Kumar, Ph.D., Massachusetts General Hospital, and Robert D. Moir, Ph.D., Massachusetts General Hospital, and Potential Order Deepak Kumar, Ph.D., Massachusetts General Hospital, and Potential Order Deepak Kumar, Ph.D., Massachusetts General Hospital, and Potential Order Deepak Kumar, Ph.D., Massachusetts General Hospital, and Potential Order Deepak Kumar, Ph.D., Massachusetts General Hospital, And Potential Order Deepak Kumar, Ph.D., Massachusetts General Hospital, Ph.D., Ph	\$150,000 ersity
The Role of Microbial Immune Responses in Alzheimer's Disease Sangram S. Sisodia, Ph.D., University of Chicago	\$250,000
Pathological Pathways and Systems	
Development of Novel APP Dimerization Inhibitors That Lower Abeta Levels Carmela R. Abraham, Ph.D., Boston University	\$100,000
Identification of Reactive Astrocyte-Secreted Neurotoxic Protein Responsible for Neuronal Apoptosis Ben Barres, M.D., Ph.D., Stanford University	\$150,000
Optimization of Pharmacologic Properties of Molecular Tweezers Gal Bitan, Ph.D., University of California, Los Angeles	\$100,000
Genetic Targets to Block Tau Propagation Marc Diamond, M.D., University of Texas Southwestern Medical Center	\$150,000
Long Abeta, Intraneuronal Amyloid and an Alternative Amyloid Hypothesis of Alzheimer's Disease Charles Glabe, Ph.D., University of California, Irvine	\$100,000
Uncovering Determinants of Neuronal Vulnerability in Alzheimer's Disease Paul Greengard, Ph.D., The Rockefeller University	\$250,000
Cell Cycle Re-entry in 3-D Human Neuron Cultures John S. Lazo, Ph.D., and George Bloom, Ph.D., University of Virginia	\$100,000
Tau Missorting in Alzheimer's Disease—Causes and Consequences Eckhard Mandelkow, Ph.D., and Eva-Maria Mandelkow, M.D., Ph.D., German Center for Neurodegenerative Diseases	\$150,000
Systemic Inflammatory Networks in Alzheimer's Disease Matthias Nahrendorf, M.D., Ph.D., and Filip Swirski, Ph.D., Massachusetts General Hospital, Harvard University	\$200,000
Regulation of RNA Translation by MAPT in Alzheimer's Disease Benjamin Wolozin, M.D., Ph.D., Boston University	\$100,000
Rejuvenation of Microglia in Brain Aging and Neurodegeneration Tony Wyss-Coray, Ph.D., Stanford University	\$150,000
Stem Cell Models	
Stem Cell Approach to Investigating the Phenomenon of Brain Insulin Resistance Sam Gandy, M.D., Ph.D., Icahn School of Medicine at Mount Sinai Hospital	\$100,000
Therapeutic Strategies	
Evaluation of AMX0035, a Neuroprotecting and M1-Deactivating Therapeutic, in an Immunological Model of Alzhein Disease (Part 1) Amylyx Pharmaceuticals Inc.	ner's \$150,000
Evaluation of AMX0035, a Neuroprotecting and M1-Deactivating Therapeutic, in an Immunological Model of Alzheimer's Disease Amylyx Pharmaceuticals Inc.	se (Part 2) \$150,000
Lead Optimization and Lead Evolution of Potent SGSMs for the Treatment of Alzheimer's Disease Steven Wagner, Ph.D., University of California, San Diego, and Rudy Tanzi, Ph.D., Massachusetts General Hospital, Harvard Unive	\$476,988 ersity
Whole Genome Sequencing and Epigenetics	
Analytical and Statistical Tools for Sequence Analysis for Alzheimer's Disease Christoph Lange, Ph.D., Harvard T.H. Chan School of Public Health	\$150,000

Total Distributed to Research

\$10,022,815

OUR SCIENTIFIC LEADERSHIP

Research Consortium

The volunteer members of Cure Alzheimer's Fund's Research Consortium develop and update our research areas of focus to identify the most promising opportunities for slowing, stopping and/or reversing Alzheimer's disease. Members pursue their own research projects consistent with these priorities and others whose work will hasten development of effective therapies for and prevention of Alzheimer's disease.



Chairman, Research Consortium Harvard Medical School/Mass. General Hospital



P. Murali Doraiswamy, M.D. **Duke University**



Karen Duff, Ph.D. Columbia University



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



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



Christian Haass, Ph.D. Ludwig-Maximilians-Universität München



David Michael Holtzman, M.D. Washington University, St. Louis



Richard L. Huganir, Ph.D. Johns Hopkins University School of Medicine



Bruce Lamb, Ph.D. Cleveland Clinic



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



Virginia M.-Y. Lee. Ph.D., M.B.A. University of Pennsylvania



Roberto Malinow, M.D., Ph.D. University of California, San Diego



Eric E. Schadt, Ph.D. Icahn School of Medicine at Mount Sinai



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



Li-Huei Tsai, Ph.D. The Picower Institute, MIT



Robert Vassar, Ph.D. Northwestern University



Steven Wagner, Ph.D. University of California, San Diego



Berislav Zlokovic, M.D., Ph.D. University of Southern California

OUR RESEARCH BENCH EXPANDED IN 2015

With 11 years of research behind us, Cure Alzheimer's Fund continues to strengthen our commitment to funding the field's most promising research. We welcomed eight new members to our scientific leadership team in 2015. Each member brings unique expertise to accelerate our progress toward a cure.

Research Consortium

- P. Murali Doraiswamy, M.D., head of the Duke University Biological Psychiatry Department
- Karen Duff, Ph.D., professor of pathology and cell biology at Columbia University
- Christian Haass, Ph.D., chair of the Metabolic Biochemistry Department at Ludwig-Maximilians-Universität München
- Bruce Lamb, Ph.D., professor, medical and molecular genetics; Roberts Family Chair in Alzheimer's Disease Research; executive director, Stark Neurosciences Research Institute, Indiana University
- Christoph Lange, Ph.D., professor of biostatistics at the Harvard T.H. Chan School of Public Health
- Li-Huei Tsai, Ph.D., director of The Picower Institute for Learning and Memory and Picower professor of neuroscience based at MIT

Scientific Advisory Board

A group independent of our Research Consortium, members of the Scientific Advisory Board advise and counsel Cure Alzheimer's Fund regarding the research priorities' overall scientific soundness. They also review individual grant proposals for consistency with the roadmap and with CAF's objectives.



Dennis Choi, M.D., Ph.D Chairman, Scientific Advisory Board Stony Brook University



Caleb Finch, Ph.D. University of Southern California



Paul Greengard, Ph.D. The Rockefeller University



Vince Groppi, Ph.D. University of Michigan



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



Robert C. Malenka, M.D., Ph.D. Stanford University



John C. Mazziotta, M.D., Ph.D. University of California, Los Angeles



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



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



Thomas C. Südhof, M.D. Stanford University

Go to **curealz.org/research/researchers** to read the full bios of all of our researchers.

Scientific Advisory Board

- Vince Groppi, Ph.D., director of the University of Michigan's Center for the Discovery of New Medicines
- Ron Petersen, M.D., Ph.D., director of the Mayo Clinic Alzheimer's Disease Research Center and the Mayo Clinic Study of Aging
- Dennis Choi, M.D., Ph.D., is now heading up our Scientific Advisory Board. Chair of the department of neurology and director of the Neurosciences Institute at the State University of New York at Stony Brook, Dr. Choi has a strong track record of research and a broad perspective on the Alzheimer's field. We welcome the insight and vision he will contribute to our research direction.

We anticipate great contributions by all of these remarkable scientists and look forward to sharing their results in future publications.

CONTINUED GROWTH

Message from the President

Tim Armour, President and CEO, Cure Alzheimer's Fund



Dear Friends.

The summary of our 2015 report could be simply, "we doubled the amount of money we put into research in 2015 from 2014!" That's a huge headline, of which we are very proud—and very grateful to all of you for making it possible.

The emphasis at Cure Alzheimer's Fund is on growth—the significant increase in the amount of money you and our Board have allowed us to "invest" in innovative research; the huge growth in contributions (compound annual growth rate of 17% for 2013 through 2015); and our organization's growing capacity to sustain and accelerate our ability to end Alzheimer's much faster than if CAF did not exist. These are all wonderful things.

But some things don't change. We are committed to the same principles now as we were when our founders started Cure Alzheimer's Fund in 2004. Succinctly, these are:

- **1.** We will focus our energy on innovative, therapy-oriented research into the causes and progression of Alzheimer's disease with the single objective of ending Alzheimer's within this generation.
- 2. We will fund the research of the world's leading Alzheimer's scientists, with a quick decision process on the basis of rolling admissions, allowing the researchers to focus on science instead of cumbersome administrative tasks.
- 3. Our Board will cover all operating costs so that all donations from everyone else will go directly to research. As our organization grows to provide more funding for research, this principle becomes a more challenging task for our Board. However, each year the Board reaffirms its commitment to this key tenet, and it has no intention of relenting.
- **4.** We will continue to be transparent about how our donors' money is spent, the nature of the research we fund and the results of that funding over time.
- **5.** We are committed to supporting collaborative research among the best Alzheimer's researchers in the world to end the disease as quickly as possible.

What's next?

The researchers Cure Alzheimer's Fund has supported over the last eleven years have made huge contributions toward ending Alzheimer's. But the disease is still with us. We are closer to ending it, but it is still claiming the "self" and lives of way too many of us. We will keep doing everything we can to accelerate the research that will contribute to this disease's demise.

With each answered question we are closer, but each answer requires more resources, more collaboration and continued commitment. The science is "bigger" now—no one researcher, lab or organization can do this alone.

But together we can.

Sincerely,

Tim Armour
President and CEO

Funding Our Vital Research

The Cure Alzheimer's Fund Board pays 100 percent of all of our operating expenses, making it possible for 100 percent of all donations to go directly to research.

CAF does not support overhead or indirect costs at recipient institutions.

CAF keeps all funds in cash equivalents. Because the objective is to move money from donors to research as quickly as possible, CAF has no endowment or investment fund.

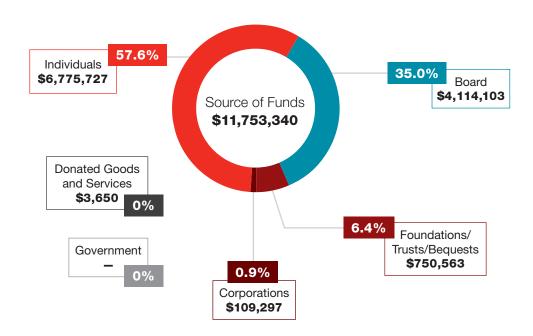
CAF funds projects approved by its Scientific Advisory Board. While proposal approval is streamlined to facilitate a focus on results rather than process, scientific integrity is CAF's top concern.

CAF has a history of "clean" audits. CAF's IRS Form 990 and audited financial statements are available online at curealz.org.

In 2015, CAF was awarded its 5th consecutive 4-star rating from Charity Navigator, the country's largest evaluator of charities. Four stars is the highest possible rating and is awarded to organizations that display sound fiscal management, accountability and transparency.

2015 FUNDRAISING

In 2015, Cure Alzheimer's Fund received financial support from 9,128 donors—individuals, corporations and foundations—totaling \$11,753,000 in cash and in-kind revenues.





the Founders and Board total \$11,949,000.

2015 FINANCIALS (Year ended Dec. 31, 2015)

Statement of Financial Position

Cash and cash equivalents \$10,034,605 Restricted cash, documentary project funds (temporarily restricted) 95,015 Contributions receivable and undeposited funds 293,115 Pledges receivable (temporarily restricted) 1,938,653 Deposits – donor-advised funds 8,997 Equipment, net 8,392 Other assets 45,948 TOTAL ASSETS 12,424,725 LIABILITIES AND NET ASSETS 166,819 Total liabilities 166,819 NET ASSETS 10,224,238 Temporarily restricted 10,224,238 Temporarily restricted 1,938,653 Documentary program project 95,015 Total temporarily restricted 2,033,668 Total net assets 12,257,906 TOTAL LIABILITIES AND NET ASSETS \$12,424,725	ASSETS	
Contributions receivable and undeposited funds 293,115 Pledges receivable (temporarily restricted) 1,938,653 Deposits – donor-advised funds 8,997 Equipment, net 8,392 Other assets 45,948 TOTAL ASSETS 12,424,725 LIABILITIES AND NET ASSETS 166,819 Total liabilities 166,819 NET ASSETS 10,224,238 Temporarily restricted 10,224,238 Temporarily restricted 1,938,653 Documentary program project 95,015 Total temporarily restricted 2,033,668 Total net assets 12,257,906	Cash and cash equivalents	\$10,034,605
Pledges receivable (temporarily restricted) 1,938,653 Deposits – donor-advised funds 8,997 Equipment, net 8,392 Other assets 45,948 TOTAL ASSETS 12,424,725 LIABILITIES AND NET ASSETS 166,819 Accounts payable and accrued expenses 166,819 Total liabilities 166,819 NET ASSETS 10,224,238 Temporarily restricted 10,33,653 Pledges receivable 1,938,653 Documentary program project 95,015 Total temporarily restricted 2,033,668 Total net assets 12,257,906	Restricted cash, documentary project funds (temporarily restricted)	95,015
Deposits – donor-advised funds 8,997 Equipment, net 8,392 Other assets 45,948 TOTAL ASSETS 12,424,725 LIABILITIES AND NET ASSETS 166,819 Accounts payable and accrued expenses 166,819 Total liabilities 166,819 NET ASSETS 10,224,238 Temporarily restricted 1,938,653 Pledges receivable 1,938,653 Documentary program project 95,015 Total temporarily restricted 2,033,668 Total net assets 12,257,906	Contributions receivable and undeposited funds	293,115
Equipment, net 8,392 Other assets 45,948 TOTAL ASSETS 12,424,725 LIABILITIES ** 166,819 Accounts payable and accrued expenses 166,819 NET ASSETS ** Unrestricted Unrestricted 10,224,238 Temporarily restricted 1,938,653 Documentary program project 95,015 Total temporarily restricted 2,033,668 Total net assets 12,257,906	Pledges receivable (temporarily restricted)	1,938,653
Other assets 45,948 TOTAL ASSETS 12,424,725 LIABILITIES 166,819 Accounts payable and accrued expenses 166,819 NET ASSETS Unrestricted Unrestricted 10,224,238 Temporarily restricted 1,938,653 Documentary program project 95,015 Total temporarily restricted 2,033,668 Total net assets 12,257,906	Deposits – donor-advised funds	8,997
TOTAL ASSETS 12,424,725 LIABILITIES AND NET ASSETS LIABILITIES Accounts payable and accrued expenses 166,819 NET ASSETS Unrestricted 10,224,238 Temporarily restricted 1,938,653 Pledges receivable 1,938,653 Documentary program project 95,015 Total temporarily restricted 2,033,668 Total net assets 12,257,906	Equipment, net	8,392
LIABILITIES AND NET ASSETS LIABILITIES 166,819 Accounts payable and accrued expenses 166,819 Total liabilities 166,819 NET ASSETS 10,224,238 Temporarily restricted 1,938,653 Pledges receivable 1,938,653 Documentary program project 95,015 Total temporarily restricted 2,033,668 Total net assets 12,257,906	Other assets	45,948
LIABILITIES Accounts payable and accrued expenses 166,819 Total liabilities 166,819 NET ASSETS 10,224,238 Unrestricted 10,224,238 Temporarily restricted 1,938,653 Pledges receivable 1,938,653 Documentary program project 95,015 Total temporarily restricted 2,033,668 Total net assets 12,257,906	TOTAL ASSETS	12,424,725
Accounts payable and accrued expenses 166,819 Total liabilities 166,819 NET ASSETS 10,224,238 Unrestricted 10,224,238 Temporarily restricted 1,938,653 Pledges receivable 1,938,653 Documentary program project 95,015 Total temporarily restricted 2,033,668 Total net assets 12,257,906	LIABILITIES AND NET ASSETS	
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NET ASSETS Unrestricted 10,224,238 Temporarily restricted 1,938,653 Pledges receivable 1,938,653 Documentary program project 95,015 Total temporarily restricted 2,033,668 Total net assets 12,257,906	Accounts payable and accrued expenses	166,819
Unrestricted 10,224,238 Temporarily restricted Pledges receivable 1,938,653 Documentary program project 95,015 Total temporarily restricted 2,033,668 Total net assets 12,257,906	Total liabilities	166,819
Temporarily restricted Pledges receivable 1,938,653 Documentary program project 95,015 Total temporarily restricted 2,033,668 Total net assets 12,257,906	NET ASSETS	
Pledges receivable1,938,653Documentary program project95,015Total temporarily restricted2,033,668Total net assets12,257,906	Unrestricted	10,224,238
Documentary program project95,015Total temporarily restricted2,033,668Total net assets12,257,906	Temporarily restricted	
Total temporarily restricted 2,033,668 Total net assets 12,257,906	Pledges receivable	1,938,653
Total net assets 12,257,906	Documentary program project	95,015
7 1 7 1 1	Total temporarily restricted	2,033,668
TOTAL LIABILITIES AND NET ASSETS \$12,424,725	Total net assets	12,257,906
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Statement of Activities

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REVENUE AND OTHER SUPPORT	
Contributions	\$10,486,909
Net assets released from restrictions (pledges)	1,250,000
Donated services	14,118
Investment income	764
Realized (loss) gain in sale of stocks	(4,202)
Unrealized gain on donor advised funds	375
Other income	3,150
Net assets released from restrictions (documentary project)	89,492
Net assets released from restrictions (Global Family Reunion project)	620,977
TOTAL REVENUE AND OTHER SUPPORT	12,461,573
EXPENDITURES	
Program expenses:	
Research distributions	10,022,663
Documentary program project expenses	89,482
Global Family Reunion project expenses	620,977
Operating program expenses	1,210,808
Total program expenses	11,943,930
Management and general	517,419
Fundraising	593,467
TOTAL EXPENDITURES	13,054,816
(DECREASE) INCREASE IN UNRESTRICTED NET ASSETS	(593,243)
TEMPORARILY RESTRICTED NET ASSETS	
Pledge contributions, net	63,478
Documentary program project contributions	80,000
Global Family Reunion project contributions	617,827
Net assets released from restrictions	(1,960,459)
(DECREASE) INCREASE IN TEMPORARILY RESTRICTED NET ASSETS	(1,199,154)
CHANGES IN NET ASSETS	(1,792,397)
NET ASSETS, beginning of year	14,050,303
NET ASSETS, end of year	\$12,257,906

From the 2015 audited financial statements which, along with IRS Form 990, are available online at curealz.org.

AWARENESS GROWS AS SUPPORT INCREASES

Alzheimer's Disease—and Cure Alzheimer's Fund—in the Spotlight in 2015

While funding the most ground breaking research is and will always be the primary focus of Cure Alzheimer's Fund, we also participate in many outreach and community events each year. In 2015, these events, coupled with an increasingly bright national spotlight on the disease, contributed to heightened Alzheimer's awareness and support for finding a cure.

From movies to music, headlines to books, daytime to primetime, Alzheimer's had an undeniable influence on American culture.



We saw Julianne Moore win a Best Actress Oscar for her beautiful and touching portraval of a woman diagnosed with early-onset Alzheimer's in "Still Alice." Cure Alzheimer's Fund and our heroes continued to leverage the film's success, and its powerful depiction of the devastation the disease causes, through screenings and book signings.

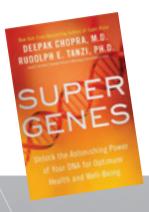
Based on the idea that music has a powerful connection to both memories and emotions, our own Rudy Tanzi collaborated with musician Chris Mann, a finalist on the hit show "The

Voice," to create the song "Remember Me." This anthem of hope, which is also now a music video, is about someone with Alzheimer's who doesn't want to be forgotten.



remember

Rudy Tanzi, chairman of the Cure Alzheimer's Fund Research Consortium, also helped keep CAF in the news through some of his own accomplishments this year. In March, Tanzi was named one of Time magazine's "100 Most Influential People" for his work on Alzheimer's in a Dish.





(from left) Dora Kovacs, Ph.D., Julianne Moore and Rudy Tanzi, Ph.D., at the TIME 100 Gala in New York City, where Tanzi was named one of the Most Influential People of 2015.

In addition, Tanzi once again collaborated with author and spiritual leader Deepak Chopra, M.D., to release their new book, "Super Genes." The book serves as a guide to help readers lead a healthier life despite the genes they inherited and take their well-being into their own hands.

TWITTER FOLLOWERS

7,300

These highly visible references to Alzheimer's in popular culture, as well as the carry-through in the media, from mainstream news outlets to Rolling Stone magazine to viral content aggregator BuzzFeed, raised awareness of the disease with new audiences.

> CNN Headline News (HLN) anchor Mike Galanos recommended Cure Alzheimer's Fund as his charity of choice for "Giving Tuesday," a global day dedicated to giving back during the holiday season. HLN ran Mike's very personal story about his father's battle with Alzheimer's several times throughout the giving season, which helped propel us to nearly triple the Giving Tuesday donations we received compared to prior years.



The first Global Family Reunion, created and spearheaded by writer A.J. Jacobs, brought together more than 3,700 people in New York City with the message that we are all "cousins" in one way or another, and that we need to fight Alzheimer's disease together. Cure Alzheimer's Fund created a "Tree of Family Memories" for the event, which enabled participants to write favorite family memories on leaves and add them to the tree.



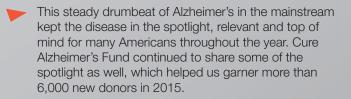
Global Family Reunion organizer A.J. Jacobs stands in front of the "Tree of Family Memories." Attendees wrote favorite family memories on leaves (below) and added them to the tree.





Sister Sledge performed "We Are Family" on the main stage.

f 71,117



OUR HEROES HAD A BUSY YEAR ... WF THANK YOU!

FIFTH-GRADE **FUNDRAISER**

Evans, Ga.

"It's great to see how these students are learning at a young age the importance of research and how contributing can help. They raised more than \$1,200. I am so proud of them."

-Teacher Britni Watts, Martinez Elementary School

RUNNING 4 ANSWERS RACE AND FUN RUN

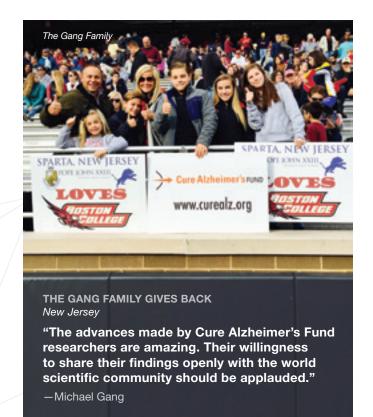
Roseland, N.J.

"Each year, I tell our participants, 'Today, while you are out on the course, think of those you love. Remember those who may not remember you. Be inspired by your own ability to do whatever you deem important enough to do."



(from left) Barbara Geiger and Carolyn Mastrangelo

-Carolyn Mastrangelo



3RD ANNUAL DICK HOLLANDER GOLF OPEN Laytonsville, Md.

"We raised a lot of funds in honor of a great man for a great cause and we know our grandfather would be proud of the work we've done. Still, we would trade it all to have him back."

-Josh Akman



\$30 FOR 30th BIRTHDAY **FUNDRAISER**

Boston

"[On my 30th birthday,] I asked my family and friends to donate \$30, or whatever they could, to fund Alzheimer's research. I wanted to raise awareness for the disease and raise money for such an important cause."

-Stefane Desmond



Stef and her dad

Thanks to all of you who have devoted your time, energy and money to supporting Cure Alzheimer's Fund. You organized races, golf and tennis tournaments, concerts, art sales, parties and more, all to raise money for Alzheimer's research. And your efforts were a huge success—together you raised more than \$323,800 in 2015!

MOM AND SON RUN OC HALF MARATHON

Long Beach, Calif.

"I am dedicated to raising awareness for this devastating disease that affects so many families, including my own."

-Ethan Lam



Ethan Lam and Kim Chan



ROTARY GOLF TOURNAMENT *Pottstown, Pa.*

"We lost my sister-in-law to Alzheimer's. We wanted to support Cure Alzheimer's Fund and we hope more Rotary Clubs will do so as well."

-Dick Cuff



Dick Cuff, past president, Bryn Mawr Rotary Club

IN 2015, HERO AND FUNDRAISING EVENTS RAISED \$323,800

PHILADELPHIA GOLF TOURNAMENT

Philadelphia

"This illness hit our family like a hurricane. I was frustrated by the ineffectiveness of the drugs my dad had been given. When I searched online for possible drugs that might be in the research pipeline, I stumbled upon



Cure Alzheimer's Philadelphia Committee (I to r): Cathy Ingham, Jim Beers, Bob DiFilippo, Tom Bond and John Coll

Cure Alzheimer's Fund's website. The next day I was on the phone with Tim Armour, president and CEO of Cure Alzheimer's Fund, asking what I could do to help."

-Cathy Ingham

A CLIMB TO REMEMBER Mont Blanc. France

"I was very impressed by the backgrounds of the founders and Research Consortium, and their track record of delivering meaningful results by way of a repeatable, private equity approach to finding a cure for Alzheimer's. I believe we all have an authentic self that is designed to do something bigger than ourselves by way of helping others less fortunate."

—Jeff Madden





IN HONOR AND IN MEMORY

Throughout the year, Cure Alzheimer's Fund receives many gifts from family and friends in honor or in memory of those with Alzheimer's disease. We are extremely grateful for everyone's generosity. It is a reminder of the extent of this terrible disease and the need to continue to fund breakthrough research.

If you would like to designate a memorial gift to Cure Alzheimer's Fund, you can do so online, by mail or by telephone. Please let us know whom to notify of your donation. We will gratefully acknowledge each gift by notifying the individuals you have designated and will do so without disclosing the amount of the donation.

Each donation will honor your loved one and help sustain our research projects.

IN 2015 PEOPLE WERE MEMORIALIZED THROUGH GIFTS IN THEIR HONOR OR MEMORY. THESE GIFTS CONTRIBUTED MORE THAN TO ALZHEIMER'S RESEARCH.

Photos sent to us are posted to the "In Memory" section of our website. To view a full listing of all memorials, please visit curealz.org/our-community.

If you have any questions about our "In Memory" program, please contact Laurel Lyle, director of fundraising programs, at Ilyle@curealz.org or 781-237-3800.

Thank you for designating charitable contributions to Cure Alzheimer's Fund.



JOIN OUR EFFORTS AND HELP MAKE A DIFFERENCE

To end Alzheimer's, Cure Alzheimer's Fund believes it is imperative to focus on and fund research that is innovative, collaborative and results oriented. The research we have funded has made tremendous advancements in understanding Alzheimer's disease and the search for a cure. But there is more work to be done.

Just over a decade ago, Alzheimer's disease was not well understood. New research was underfunded and researchers were not encouraged to think big or bold about how to tackle it. Years of drug development investments by pharmaceutical companies had not yielded a therapeutic solution. Research on Alzheimer's disease lacked support and momentum.

Cure Alzheimer's Fund has worked to reverse the situation. Frustrated by the slow pace of research—and leveraging their experience in venture capital and corporate start-ups—our founders, Henry McCance, Phyllis Rappaport, and Jacqui and Jeff Morby, came together in 2004 to build a new Alzheimer's research fund designed to dramatically accelerate research, make bold bets and find a cure.

We invite you to join our ongoing efforts to support the most promising and productive research to end Alzheimer's disease. We are targeting truly breakthrough work that is accelerating the efforts to reach a cure.

To make a gift, or for more giving information, please visit our website: curealz.org/donate.

Or call: 877-CURE-ALZ (287-3259).



CHARITY DESIGNATION

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 #52-239-6428.









OUR BOARD AND STAFF

Cure Alzheimer's Fund is governed by a board of directors; administered by a small, full-time staff; and guided scientifically by a Research Consortium. A Scientific Advisory Board audits the research program to make sure it is consistent with the objectives of the foundation. Go to curealz.org/about/people to read the full bios of all of our board members and staff.

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MISSION

To fund research with the highest probability of preventing, slowing or reversing Alzheimer's disease.

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