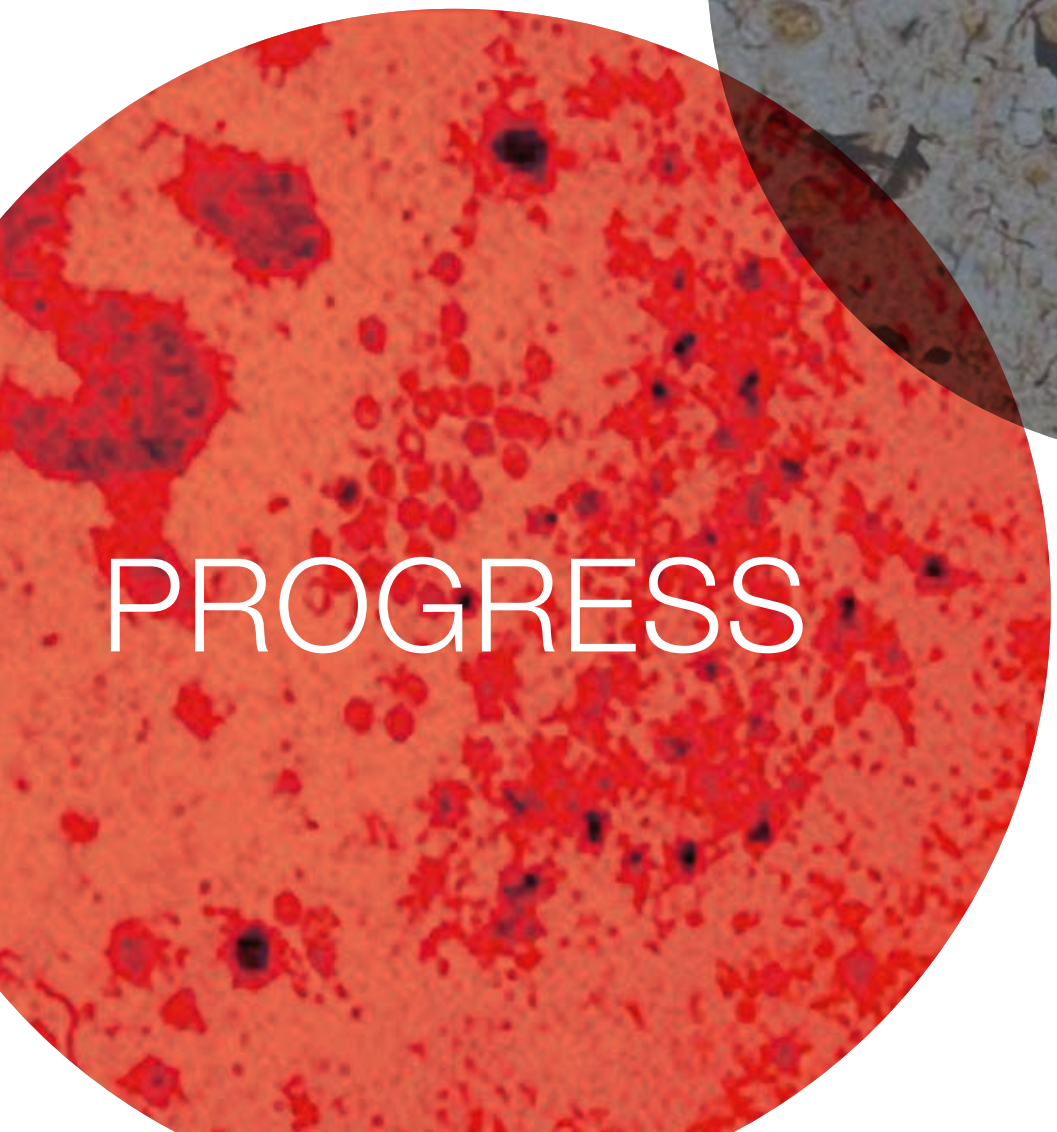


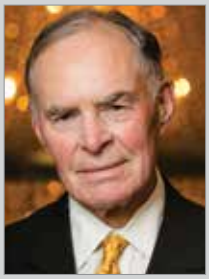
Cure Alzheimer's FUND

ANNUAL REPORT 2013

2013 ANNUAL REPORT

ON OUR COVER: The themes of collaboration, persistence and progress typify our efforts in 2013. Here they are depicted as three overlaying circles portraying the progression of Alzheimer's disease pathology through amyloid, tau and inflammation, which represent key points of intervention (see our Alzheimer's Disease Model on page 7).

COLLABORATING FOR A CURE



Message from the Chairman

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

Dear Friends,

We have at last achieved an integrated understanding of the causes of Alzheimer's disease and have created a scientific infrastructure and processes that are allowing us to understand and address most, if not all, of Alzheimer's multifaceted causes. As a result, we have entered into a highly productive stage of our research, in which we now are in a position to define with some precision "intervention points" at which we can target specific potential scientific interventions focused on different causes of the disease (see page 7).

Dr. Tanzi has remarked to me he is as excited about this facet of our evolution as he was more than 15 years ago when he co-discovered the first three Alzheimer's genes. In his commentary, which follows, Rudy will provide you with a discussion of the state-of-the-art projects he oversees as chairman of our Research Consortium. But below, let me provide you with an overview from my perspective.

In terms of immediacy, our first potential cure/preventive will shortly be on its way to human trials—a statin-type drug for Alzheimer's disease.

A gamma secretase modulator (GSM) is a therapy for modulating (e.g., safely reducing) the production of Abeta in the brain. We have been funding the development of such a preventive for some time, and the National Institutes of Health (NIH) now has become so enthused it is "fast tracking" the project and pouring millions of its own money into refining the product in preparation for human trials this year. The GSM is a drug that would be taken by mouth, with the dosage dependent on Abeta concentrations in the brain—in much the same way that statin dosage is determined by cholesterol levels.

We have developed the first "Alzheimer's Model," which defines the principal causal dimensions of the disease and allows us to identify "intervention points" to which research is directed.

As the reader will note from the diagram on page 7, Alzheimer's disease pathology arises in different stages in the life of an individual, usually beginning early with the excessive creation of Abeta proteins in the brain, the impact of which does not show up until later in life. If one can moderate that process, it is likely that Alzheimer's pathology will not develop. Therefore, one early-stage intervention strategy is to be able to moderate that process, and that is the purpose of the GSM described above (as well as other projects under way).

However, as Alzheimer's progresses, there are a variety of intervention points at which research projects can be aimed to avoid the evolution of Alzheimer's pathologies at those stages. As described in the body of this report, our research is focused on a number of these intervention points. The research interventions are designed (among other things) to develop means to:

a. prevent the accumulation of Abeta by lowering production or enhancing clearance;

b. prevent Abeta from damaging brain cells and synapses;

c. prevent the generation of tau tangles by Abeta or other factors;

d. prevent the spread of tau tangles within the brain;

e. prevent inflammation within the brain; and

f. speed or accelerate the clearance from the brain of Abeta and other toxic substances.

Our Scientific Infrastructure

One of the reasons we think we are at a new stage of evolution is that we now have a complete scientific infrastructure, which will allow us to greatly accelerate our progress. Some of the elements of that infrastructure are:

Databases of the genetics and biology of Alzheimer's disease

The accumulated information includes results from the first genome-wide association screen for Alzheimer's disease genes started in 2005, and the first **Whole Genome Sequencing (WGS)** of Alzheimer's disease families, begun in 2012. In addition, our **AlzGene database**, updated continually, available free of charge to all scientists and containing systematic information on all Alzheimer's-related genetic research from all over the world, now is being completely overhauled to include whole genome sequencing information as well.

This collection of databases, particularly that of the WGS, has permitted us in an extremely short time to identify nearly 1,000 new genetic mutations in more than 50 different AD genes. Perhaps most importantly, Dr. Tanzi and his team have been able to identify the conceptual linchpin (key causal agent) that creates the environment enabling other causal agents to become "bad actors" in the proliferation of Alzheimer's pathology. As Dr. Tanzi will explain, that linchpin is the brain's innate immune system.

A Powerful Stem Cell Consortium

Under the leadership of Dr. Sam Gandy of the Icahn School of Medicine at Mount Sinai, we have been able to put together one of the most powerful groups of stem cell institutions in the world. The Cure Alzheimer's Fund Stem Cell Consortium focuses on a variety of opportunities for creating stem cells from normal tissue, using those cells for gaining insights into the nature of neurogenesis in the brain, developing new scientific applications of such knowledge, and using stem cells created from Alzheimer's patients to analyze the pathology of Alzheimer's disease and potentially to heal cell damage in the brains of Alzheimer's patients.

Totally new advanced research tools

To take full advantage of the new technologies described above, it has been necessary to create new research tools. A few of these tools are as follows:

a. Alzheimer's in a dish: Unbelievably, the stem cell researchers in Dr. Tanzi's lab have been able to create an amazing new technology: "AD in a dish." The dish in this instance is a miniature container of nerve cells produced from human stems cells with Alzheimer's mutations that produce the pathology of the disease: senile plaques, Abeta oligomers and tau tangles. It is the first true recapitulation of Alzheimer's disease brain pathology in a Petri dish, in which researchers now can better understand the disease process and rapidly test new potential drugs, etc. It is a tremendous research tool, since these "mini-brains" can be created quickly for a variety of tests—much cheaper and faster than using transgenic mice for testing. Furthermore, the system can be customized to one's own personal genetics, as each new drug is tested to get an idea of whether a specific drug will work in that individual.

b. Proprietary algorithms to be used to analyze and manipulate the "big data" generated as a result of the WGS project: As a result of a $4 million grant from the National Institute of Mental Health, our researchers have been able to develop truly advanced mathematical

More than 2,500 new families chose to support us this year, bringing our funding to record levels (and as usual, the founding families and directors paid all operating costs so that third-party donations could go 100 percent into research).

and statistical tools for analyzing the data contained in the WGS database. This essentially is a highly advanced type of bioinformatic analysis system, which has to be used when analyzing quadrillions of bytes of genomic information (petabytes) in a biological context. With these tools it is possible to analyze DNA sequences down to a single base or base pair (of which there are 6 billion in the human genome) and to view sequences of bases and see the relationships between different segments or repeat segments. These algorithms are allowing us to identify countless genetic variants never before discovered and understand how they affect human physiology.

c. **Screening currently available drugs against our database of Alzheimer's genes:** There currently are thousands of existing and approved drugs used for a great variety of human illness. Some of these could have application to Alzheimer's disease and may offer the possibility of protection against the disease. If such drugs were found and tested, this could advance the pace to a cure dramatically, since FDA testing and approval processes would be rapid, and the drugs could be brought to market quickly. As Rudy will explain, we now are in the process of creating another large database in which approved drugs are matched against our AD gene database to identify those drugs that possibly may have some impact on the Alzheimer's genes.

Thanks and Congratulations

Finally, this letter would not be complete without thanks and congratulations from our founders and directors, our staff and our scientists.

We received resounding support from all of our old and new friends this year. More than 2,500 new families chose to support us this year, bringing our funding to record levels (and as usual, the founding families and directors paid all operating costs so that third-party donations could go 100 percent into research).

The funds you provide through your generosity, in addition to supporting research, also are leveraged by others. CAF is an innovator and risk taker. Once we demonstrate "proof of concept," others are happy to join with us to fully exploit whatever innovation we have originated, either in the form of direct funding or co-funding of joint projects. So your funds go a long way, as shown on page 30.

The innovations just described would not be possible without the brilliance of our researchers. A hearty thanks goes out to all of them for their outstanding work this year. And particular, thanks and "welcome" go to our new members of the Research Consortium and Scientific Advisory Board:

Richard Huganir, Ph.D.;

William Mobley, M.D., Ph.D.;

Eric Schadt, Ph.D.; and

Marc Tessier-Lavigne, Ph.D.

Finally, our congratulations go to Thomas Südhof, M.D., a member of our Scientific Advisory Board, who was honored in 2013 with the award of the Nobel Prize in Physiology or Medicine. Congratulations, Tom.

Nine years since the founding of Cure Alzheimer's Fund have passed quickly. All of you have made CAF what it is. I personally thank you for all of your support over the years.

Jeffrey L. Morby
Chairman and Co-Founder

PERSISTENCE IN RESEARCH

Message from the Research Consortium Chairman

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

Dear Cure Alzheimer's Fund Supporters,

As I reflect on the research progress made in 2013, I not only am struck by how much the Cure Alzheimer's Fund community has accomplished, but also how different our knowledge about Alzheimer's and our progress toward successful treatment would be if there were no Cure Alzheimer's Fund community. My research colleagues and I are deeply grateful for the support we have received that has enabled this work, which without Cure Alzheimer's Fund would not have been possible. Now, let me share with you the highlights of what we have accomplished together.

Innate Immunity is Key

Thanks to the discovery of a host of new Alzheimer's genes, we have learned that innate immunity and the genes that regulate it in the brain are the keys to understanding how the majority of neurons die from inflammation. Innate immunity is the process by which the body and brain are protected from infections owing to microbial pathogens like bacteria, viruses and yeast. Our seminal discovery of the association of Alzheimer's disease with the CD33 gene in 2008 and, more recently, its relation to the Alzheimer's-associated TREM2 gene (in 2012) has taught us an immense amount about how our brains defend against pathogens. This same process of defense also can be stimulated by rampant Alzheimer's pathology in the brain—in the form of senile plaques and nerve cell tangles.

We now have found that the CD33 and TREM2 genes work together to regulate microglial cells and inflammation in the Alzheimer's brain. Normally, microglial cells help to nourish nerve cells. But when they receive signals that the brain is under attack, e.g., by bacterial infection, they become soldierlike and try to kill the pathogens. Abundant Alzheimer's pathology and nerve cell death also can trigger microglial cells to become soldiers shooting oxygen-based bullets called free radicals. While meant to protect the brain, the activated microglial cells inadvertently can kill nerve cells as part of "friendly fire" aimed at microbial pathogens. In this case, there are no microbial pathogens, just plaques, tangles and dying nerve cells that trick the brain into this neuroinflammatory state. This is arguably one of the biggest findings in the field in the last several years. In fact, Alzforum, the most authoritative online publication regarding Alzheimer's research, listed our new genes in two of its just-released top five trends in Alzheimer's for 2013. The Alzforum also listed our discovery of the late-onset AD mutations in the ADAM10 gene, which would not have been possible without Cure Alzheimer's Fund support. Because of leverage from our early investment, we have federal grants to expand all of these programs.

We now have discovered the very first new early-onset familial AD genes to be discovered since we and others first co-discovered the presenilin genes in 1995.

Alzheimer's Genome Project™ (AGP)

We now have discovered the very first new early-onset familial AD genes to be discovered since we and others first co-discovered the presenilin genes in 1995. This was largely attributable to our AGP Phase 2 results and a revolutionary new algorithm, developed in my lab, for finding structural DNA variants that segregate with disease in families. We were able to report 10 new AD genes with early-onset AD-linked mutations consisting of large insertions, deletions and rearrangements of the DNA in these genes.

Once again this year, *TIME* magazine listed the discovery of new AD genes in its top 10 medical breakthroughs. The piece generally referred to all new AD gene studies, not just a single paper, and specifically mentions "innate immune responses and inflammation," which has been championed by us thanks to Cure Alzheimer's Fund support!

Whole Genome Sequencing

We now have found many new mutations in APOE that may influence whether APOE4 leads to AD or not. We also have found 85,000 new mutations and DNA variants in and around the new AD genes that we and others have identified over the last several years. Of these, we have identified hundreds of new pathogenic mutations in virtually all of the new AD genes previously identified in the Alzheimer's Genome Project™ and similar projects. We also now have begun a series of very exciting collaborations with world-renowned bioinformatics expert and new member of our Research Consortium Eric Schadt (Icahn School of Medicine at Mount Sinai), along with Win Hide (head of our bioinformatics subgroup; Harvard School of Public Health), to determine what these new genes and their mutations can tell us about a.) relevant biological and physiological pathways in AD that previously have not been implicated), and b.) what can these genes, when considered together as a group, tell us about currently approved drugs that may be useful for AD—those that never would have been considered until all of these genes were analyzed together in pathway- and systems-based analyses. As mentioned above, we are using the new gene mutations found with whole genome sequencing to make new neuronal and animal-based disease models and develop new therapies. So far, once again, innate immunity has emerged as a key event in Alzheimer's pathology. As such, we are rapidly ramping up to develop therapies that will regulate brain neuroinflammation subsequent to the accumulation of Abeta, nerve cell tangles and nerve cell death in the brains of patients.

Gamma Secretase Modulators: Moving Toward Clinical Trials

Steve Wagner, Ph.D., Research Consortium member and professor at the University of California, San Diego, and I have developed drugs aimed at regulating the activity of an enzyme called gamma secretase, which is a critical contributor to Abeta production. This effort has been so successful the compounds we have developed have been adopted by the National Institutes of Health (NIH) as part of its fast-track, high-priority "Blueprint" program. We have some of the most promising gamma secretase modulators in the world, capable of lowering production of Abeta, safely, without adverse "off-target" effects. The NIH Steering Committee reviewed our drugs in November 2013 and gave us the thumbs up to continue and to aim at having a candidate go to the FDA for approval for clinical trials over the coming year. This is a huge achievement, especially for academic labs—all made possible by Cure Alzheimer's funding.

Collaborative Work By Our Research Consortium and Scientific Advisory Board Members

If I had to point to one particular theme for 2013, it clearly would be "collaboration." Our Research Consortium members have been working together with each other and their collaborators with increasing frequency and alacrity. We have exciting collaborations that have emerged by bringing together some of the greatest minds in the field for our Research Consortium (RC); they speak, email and meet on a regular basis and with increasing frequency. Dozens of high-impact papers have come out of these collaborations that otherwise would not have existed. For example, under the leadership of Sam Gandy at the Icahn School of Medicine at Mount Sinai, we have put together arguably the most powerful stem cell consortium in Alzheimer's research (see pages 6 and 14. Exciting studies on our new Alzheimer's genes are now in progress with Paul Greengard, Nobel Laureate at The Rockefeller University; Sam Sisodia, University of Chicago; John Hardy, Brain Research Trust; Allan Levey, Emory University; Roberto Malinow, University of California, San Diego; Gandy; Betza Zlokovic, University of Southern California; and Robert Vassar, Northwestern University, to name a few. See page 23 for a complete list of our Research Consortium and Scientific Advisory Board Members.

Cure Alzheimer's Fund Stem Cell Consortium (CAFSCC)

The Cure Alzheimer's Fund Stem Cell Consortium (CAFSCC) was founded in 2013 as a collaborative program involving laboratories at the Icahn School of Medicine at Mount Sinai (ISMMS), New York Stem Cell Foundation (NYSCF), Harvard Medical School (HMS), Massachusetts General Hospital (MGH); The Rockefeller University (RU); and Hadassah University (HU). The labs were chosen for their complementary skills and areas of focus, and these choices already have begun to yield dividends in terms of collaborative publications and collaborative grants elucidating the process of how Alzheimer's disease begins and progresses.

The CAFSCC, within the space of one year, has developed highly productive collaborations wherein each lab employs its particular expertise to build one of the various technological way stations that mark the "bench-to-bedside" pathway, culminating in a trajectory that, within a short time, should yield information relevant to personalized pharmacogenomics recommendations in subjects with the most common deterministic cause of hereditary AD.

The Disease Model

The disease model of Alzheimer's pathology (see page 7) is an outstanding example of the output of collaboration and progress of our Research Consortium. The model was produced by all of the members of the Research Consortium as a way to depict the current (2013) understanding of how Alzheimer's starts and progresses through the brain, and indicates where the pathology is most vulnerable to intervention. The model shows essentially a vicious cycle of destruction of brain cells or neurons beginning with the overproduction of the protein Abeta, which stimulates the spreading of another protein called tau that "infects" other cells, causing inflammation in the brain. This inflammation stimulates the brain's defenses to react in a way that creates more Abeta and tangles. We also have learned that while Alzheimer's usually begins with the accumulation of Abeta, it also can start with either tangles or inflammation, e.g., subsequent to traumatic brain injury, as part of one vicious cycle. Each of these major transitions—creation of Abeta, creation and spreading of tau tangles and brain inflammation—offers clear opportunities for therapeutic intervention. It would have been impossible to get leading Alzheimer's researchers to agree to such a depiction until very recently. But the work done by Cure Alzheimer's Fund researchers and others has brought Alzheimer's research to the point where most investigators agree on this broad outline. The details are many, complex and still unfolding, but this is strong indication of the accelerated pace toward the development of truly effective therapies to stop the disease before it starts, interrupt its development in the early stages and retard or perhaps even reverse its effects in those who already are symptomatic. Much work is left to be done, but the objectives and even some of the pathways to achieve the objectives now are much clearer.

The Science of Cure Alzheimer's

These are just some of the highlights from 2013 alone. See the complete list of 2013 projects on page 9 for an idea of the depth and scope of the research made possible by CAF, all of which is moving us more rapidly to our goal. The science of Cure Alzheimer's Fund is now a powerful and well-oiled machine, moving so fast and efficiently that it is a challenge to keep up with the new discoveries, papers, grants and collaborations that are emerging in the CAF nexus. Emails concerning collaborations constantly are being sent among Research Consortium members and their team members. We are writing grants together. There is so much going on—the research we fund is exploding with results.

In closing, I can say these are the most exciting and productive days of my scientific career, largely thanks to Cure Alzheimer's Fund. My goal, and that of my research colleagues, is to stop this disease. And as all of this tremendous collaboration and progress shows, we cannot do it without you.

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

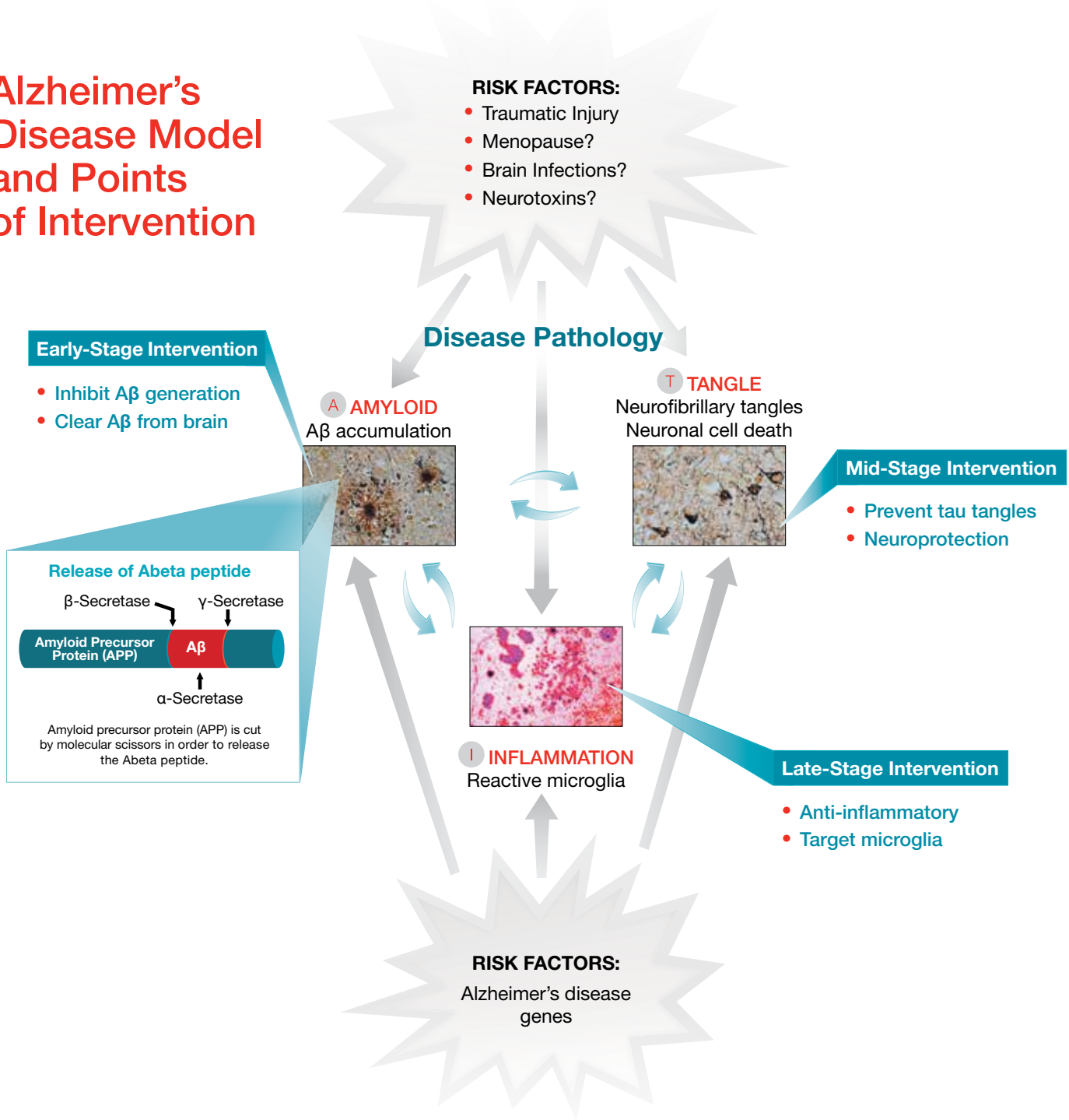
Alzheimer's: How to Intervene

In recent years, a scientific consensus has started to emerge about how Alzheimer's disease originates and develops in a vicious cycle of Abeta peptide accumulation, nerve cell death and inflammation, followed by even more nerve cell death. Research sponsored by Cure Alzheimer's Fund has provided substantial contributions to this understanding—including the identification of key genes and the functions of those genes. This has allowed the entire research community to focus on more targeted intervention.

This model gives a simplified illustration of the emerging consensus and the three basic strategies for intervention:

- An early-phase intervention, inhibiting the production of the toxic Abeta peptide, and/or clearing it from the brain after it forms.
- A mid-phase intervention that would inhibit the formation of tau tangles and protect neurons from undue stress.
- A late-phase intervention that would fight inflammation and thus slow down or even stop the disease process.

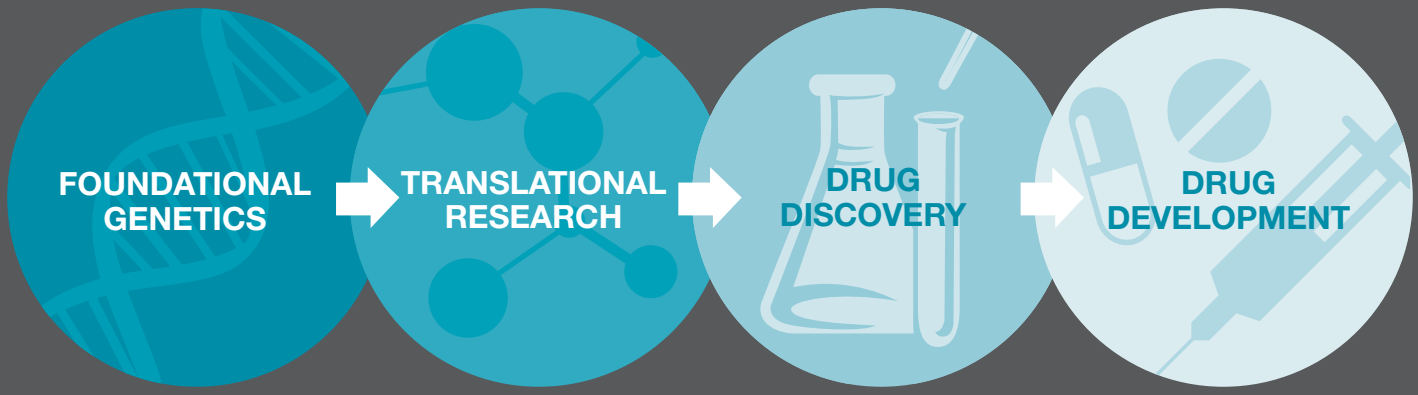
Alzheimer's Disease Model and Points of Intervention



RESEARCH ROADMAP

Cure Alzheimer's Fund is the only research organization with a real plan to end Alzheimer's disease. Our unique funding strategy is 100 percent focused on finding a cure and supports the scientists doing the most innovative work to move knowledge of Alzheimer's pathology most expeditiously to prevention and cure. Our ultimate objective is to stop the disease before it even starts.

By addressing the problem at the root and finding the major causes of the disease, we already are accelerating developments of effective therapies. The projects we fund are based on our roadmap, which we think is the quickest way to a cure.



Find all genes that contribute to risk 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 identify 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

2013 RESEARCH PROJECTS

In 2013, Cure Alzheimer's Fund distributed $4,576,000 for research supporting 14 projects.

Roadmap	Intervention Point*	Project	Researcher
	A	**The Amylin Protein of Diabetes Mellitus is an Antimicrobial Peptide**	Robert Moir, Ph.D. Rudy Tanzi, Ph.D.
	A T I	**Alzheimer's Genome Project™: Phases II and III**	Rudy Tanzi, Ph.D.
	A	**Characterization of the Pathological Significance of a Novel Type of Vascular Amyloid**	Charles Glabe, Ph.D.
	A	**The Root of Alzheimer's Disease: Purification and Characterization of Amyloid-beta Oligomers from the Human Brain**	David Brody, M.D., Ph.D.
	A	**BACE1 Transcytosis in Alzheimer's Disease Pathogenesis, Year 2**	Gopal Thinakaran, Ph.D.
	A	**The Roles of Eps Homology Domain (EHD) Proteins and Synaptic Activity in Axon Transport of the Alzheimer's Beta-secretase BACE1 in the Brain, Year 2**	Robert Vassar, Ph.D.
	A T	**Stem Cell Consortium**	Doo Yeon Kim, Ph.D. Scott Noggle, Ph.D. Sam Gandy, M.D., Ph.D. Tamir Ben-Hur, M.D., Ph.D.
	A T	**Abeta Oligomers and the Pathogenic Spread of Tau Aggregation: Implications for Alzheimer's Disease Mechanism and Treatment**	Dominic Walsh, Ph.D. Dennis Selkoe, M.D.
	T	**Sleep and Tauopathies: Effect of an Anti-Tau Antibody**	David Michael Holtzman, M.D.
	I	**Vascular Regenerative Therapy for Alzheimer's Disease**	Guojun Bu, Ph.D.
	n/a	**Discovery of Alzheimer's Disease Blood Biomarkers Using Phage Display Technology**	Yueming Li, Ph.D.
	A	**Effects of Inhibitors of Monoacylglycerol Lipase on Behavior and Synaptic Plasticity of Ts65Dn Mice, a Genetic Model of Down Syndrome**	William Mobley, M.D., Ph.D.
	A	**Elucidation of the Mechanism of Action of Gamma Secretase Modulators**	Steven L. Wagner, Ph.D.
	A T	**Normalizing Abeta Synaptic Depression with Drugs Targeting PICK1**	Roberto Malinow, M.D., Ph.D.

Alzheimer's Disease Model and Points of Intervention

A **AMYLOID -** Abeta accumulation

T **TANGLE -** Neurofibrillary tangles and neuronal cell death

I **INFLAMMATION -** Reactive microglia

* See the full Disease Model and Points of Intervention on page 7.

The Amylin Protein of Diabetes Mellitus is an Antimicrobial Peptide

A

March 2013

$300,000

Robert Moir, Ph.D.

Rudy Tanzi, Ph.D.

Harvard Medical School/
Massachusetts General Hospital

The goal of this project is to determine whether the amylin (IAPP) protein has a role in innate immunity (similar to Abeta) in order to significantly advance our understanding of the origins of diabetes pathology and its possible linkage to Alzheimer's disease.

The underlying cause of Type 2 diabetes mellitus remains unclear. In 1987, researchers found an important clue to the pathological mechanisms underpinning the disease—insoluble deposits of a small protein called amylin (IAPP) that form in pancreatic islets of those with diabetes. Proteinaceous deposits of this kind are known as amyloid and are a pathological hallmark of a number of common diseases, including Alzheimer's disease (AD). Different amyloid-forming proteins are associated with different diseases. However, amyloid-forming proteins often share physiochemical properties, and their associated diseases have overlapping pathologies. The similarities between IAPP and Abeta are particularly striking. Abeta is present in the brains and pancreatic islets of patients with diabetes. Both IAPP and Abeta are small, amphipathic molecules generated by cleavage of larger membrane-associated precursor proteins and bind the molecular chaperone apolipoprotein E. Abeta and IAPP also share another important similarity—despite two decades of intensive study, the normal nonpathogenic functions of these proteins are poorly understood.

Our laboratory recently advanced the novel idea that Abeta is part of the innate immune system and belongs to a family of proteins called antimicrobial peptides (AMPs). AMPs function as natural antibiotics to protect against invading pathogens. *In vitro*, Abeta can inhibit the growth of at least eight clinically important pathogens. In addition, homogenates prepared from the brains of AD patients have specific Abeta-mediated antimicrobial activity. Preliminary data from our latest experiments show IAPP also has antimicrobial activity and inhibits the growth of the important human pathogens *Candida albicans* and Listeria monocytogenes. In initial tests, IAPP antimicrobial activity was equivalent to Abeta, although the peptide may target a narrower microbial spectrum.

Our discovery of Abeta's role in immunity identifies pharmacological manipulation of the innate immune system as a new and promising therapeutic strategy for treating AD. Strong epidemiologic evidence suggests an association between AD and Type 2 diabetes, but the critical pathological mechanism common to both diseases has yet to be identified. Our preliminary findings link, for the first time, the amyloid-forming proteins of these two disorders with a common nonpathological function as innate immune effector molecules. We propose a project to investigate IAPP for a role in innate immunity using an experimental paradigm similar to that used in the study of Abeta. We think findings from this new line of inquiry may significantly advance our understanding of the origins of diabetes pathology and potentially form the basis for a new therapeutic strategy for curbing the rising diabetes epidemic.

Alzheimer's Genome Project™

Phases II and III of the Alzheimer's Genome Project are being carried out in parallel.

A T I

Phase II

February 2013

$1,200,000

Rudy Tanzi, Ph.D.

Harvard Medical School/ Massachusetts General Hospital

The goal of this project is to evaluate our new Alzheimer's disease gene candidates for effects on Alzheimer's pathology and related biological pathways, including APP processing, Abeta protein generation, tangle formation and cell death. These studies are being carried out as part of Phase II of the Alzheimer's Genome Project™ (AGP) and entail functional analyses of the Alzheimer's gene candidates identified in Phase I of the AGP. We have focused the Phase II studies on the novel Alzheimer's genes known as ADAM10, ATXN1 and CD33, all identified in 2008 as part of Phase I of the AGP.

The functional studies, aimed at how these genes influence risk for Alzheimer's, are carried out in both cell-based and animal models. We also have performed genetic follow-up and functional studies for AD-associated aberrations in the human genome, known as copy number variants (CNV). This has led to the identification of several CNVs in novel Alzheimer's genes underlying the inheritance of cases of familial early-onset Alzheimer's that were not explained by the known early-onset Alzheimer's genes co-discovered by our lab in the 1980s and '90s (amyloid precursor protein, presenilin 1 and presenilin 2).

The knowledge gained from how the newly identified Alzheimer's genes (from Phase I) biologically increase or decrease risk for Alzheimer's disease is being implemented to design new drug discovery efforts, also as part of Phase II of the AGP. Phase III of the AGP is being carried out parallel to Phase II and includes Whole Genome Sequencing of the human genomes of subjects from both early-onset and late-onset Alzheimer's families. The goal of Phase III of the AGP is to identify all of the biologically relevant functional gene variants that influence risk for Alzheimer's disease. Once identified, these gene variants will be analyzed using similar methods to those described here in Phase II of the AGP.

Phase III

April 2013

$1,500,000

We will carry out Whole Genome Sequencing (WGS) of all subjects in the National Institute of Mental Health (NIMH) Alzheimer's disease family sample (1,510 subjects; 437 AD families). We will identify functional DNA variants throughout the human genome that are inherited as risk factors for Alzheimer's disease. We also will analyze DNA from brain samples of subjects who exhibited significant Alzheimer's pathology at autopsy, but never suffered from dementia; this will allow us to identify protective gene variants as well.

This study constitutes Phase III of the Alzheimer's Genome Project™. While Phase I and II informed regarding which genes are implicated in risk for Alzheimer's disease, this study will allow us to assess the entire human genome, including the 96 percent that is not made up of "genes," per se, but instead includes the DNA that regulates the activity of the genes. While the goal of Phases I and II was to identify all of the genes involved in Alzheimer's disease susceptibility, in Phase III, we will (1) determine all of the DNA variants in the Alzheimer's genes that directly influence risk for the disease; and (2) determine all of the DNA variants in the intergenic portions of the genome that regulate the activities of the Alzheimer's genes.

As in the past, we will use this information to determine exactly how each Alzheimer's gene (emerging from Phase I and II) functionally affects risk for the disease at the biological level. These findings then will be used not only to better understand the causes of Alzheimer's disease, but also to guide drug discovery efforts to slow down, stop or, perhaps, even reverse the disease process.

Characterization of the Pathological Significance of a Novel Type of Vascular Amyloid

A

October 2013

$100,000

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

The amyloid Abeta peptide is deposited in at least two distinct locations in the AD brain: Parenchymal plaques and vascular amyloid deposits in the wall of arterioles, where it is associated with vascular smooth muscle cell degeneration and stroke (Congophilic amyloid angiopathy, CAA). While CAA is commonly found in the AD brain, some human mutations within the Abeta domain of the amyloid precursor protein (APP) cause CAA and stroke rather than AD, indicating that these diseases can occur independently. Using a conformation-dependent monoclonal antibody, M31, we have discovered a structurally unique type of Abeta deposit that is specifically associated with vessels. This shows that a subset of the vascular amyloid is conformationally unique and raises the hypothesis that it may be associated with a unique type of pathogenesis.

The goal of this proposal is to examine the relationship of this unique vascular amyloid to AD and CAA pathogenesis and obtain preliminary data to support an NIH application with more mechanistic and translational aims. The results of this study may lead to the development of immunological strategies to therapeutically target CAA and image its accumulation in the human brain, allowing the pre-mortem diagnosis of vascular amyloidosis and the stratification of patients for human clinical trials for both AD and CAA.

The Root of Alzheimer's Disease: Purification and Characterization of Amyloid-beta Oligomers from the Human Brain

A

November 2013

$100,000

David Brody, M.D., Ph.D.
Washington University
School of Medicine

Large, poorly soluble aggregates of the Abeta peptide form the senile plaques that are a pathological hallmark of Alzheimer's disease, but the extent of plaque deposition correlates only moderately with dementia; for example, many middle-aged and elderly people have extensive plaque deposition without any signs of dementia. Instead, several types of smaller, water-soluble Abeta oligomers have been found to be more toxic than either plaques or Abeta peptide monomers. Our collaborative group has recently developed a sensitive, specific, quantitative and high-throughput assay for Abeta oligomers. We propose to use this assay to facilitate purification of Abeta oligomers from human brain tissue. We expect there will be substantial complexity in the Alzheimer's disease brain, with multiple oligomeric species having varying structural properties and toxicities. Once purified, we will use mass spectrometry to characterize the structure and cell-based toxicity assays to quantitatively assess the function of each distinct type of oligomer. The first major outcome will be identification of critical post-translational modifications, associated proteins and conformational epitopes in Abeta oligomers that could be targeted by innovative therapeutics. The second major outcome will be determining which (if any) animal models accurately reproduce the Abeta oligomers found in the human brain so that candidate therapeutics targeting these oligomers can be tested appropriately *in vivo*. It may be that entirely new animal models will be needed. Thus, if successful, this project will facilitate an entirely new wave of preclinical and clinical therapeutic development for Alzheimer's disease.

BACE1 Transcytosis in Alzheimer's Disease Pathogenesis, Year 2

A

December 2013

$100,000

Gopal Thinakaran, Ph.D.
University of Chicago

In Year 2 of Cure Alzheimer's Fund funding, we will perform proof of concept experiments to validate EHD proteins and Rab11 as viable AD therapeutic targets in the brain. Year 1 experiments mainly utilized neurons grown in a dish; therefore, it is critical to verify that EHDs and Rab11 control BACE1 axonal trafficking and sorting and Abeta production in neurons in the context of the adult brain. For these experiments, we will use two different genetically engineered mouse models, called BACE1-YFP and PDAPP mice, the former of which we generated specifically for this project. These essential validation experiments are necessary before we can undertake future drug development efforts to target EHDs and Rab11 for AD.

In summary, the current proposals represent drug target identification and validation projects that we plan to take into drug discovery efforts in the future. In Year 1 we have identified EHD proteins and Rab11 as potential therapeutic targets for AD. In Year 2 we will perform proof of concept experiments to establish a model for validating other endosomal pathway genes identified by Dr. Tanzi's Whole Genome Sequencing screen of AD risk factor genes.

The Roles of Eps Homology Domain (EHD) Proteins and Synaptic Activity in Axon Transport of the Alzheimer's Beta-secretase BACE1 in the Brain, Year 2

A

December 2013

$100,000

Robert Vassar, Ph.D.
Feinberg School of Medicine

See abstract above.

Stem Cell Consortium

A T

January 2013

$400,000

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

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

Doo Yeon Kim, Ph.D.
Harvard Medical School/
Massachusetts
General Hospital

Scott Noggle, Ph.D.
New York Stem
Cell Foundation

The Cure Alzheimer's Fund Stem Cell Consortium (CAFSCC) was founded in 2013 as a collaborative program involving laboratories at the Icahn School of Medicine at Mount Sinai (ISMMS; Gandy), New York Stem Cell Foundation (NYSCF; Noggle), Harvard Medical School (HMS; Eggan), Massachusetts General Hospital (MGH; Kim); The Rockefeller University (RU; Tessier-Lavigne); and Hadassah University (HU; Ben-Hur).

The labs were chosen for their complementary skills and areas of focus, and these choices already have begun to yield dividends in terms of collaborative publications and collaborative grants. The projects are discussed below in a sequence related to one narrative of how Alzheimer's disease (AD) begins and progresses:

Genes known to play deterministic or risk-enhancing roles in major neurodegenerative diseases include presenilin 1 (PS1), presenilin 2 (PS2) and microtubule-associated protein tau (MAPT). The Gandy, Noggle and Tessier-Lavigne labs are focusing on generation of induced pluripotent stem cells (iPSCs) from fibroblasts taken from patients with pathogenic mutations or high-risk polymorphisms. An initial paper on iPSCs derived from subjects with PS1 mutations was published in the journal *Public Library of Science ONE* (*PLoS ONE*) on Jan. 8, 2014. This paper describes the production of iPSCs and iPSC-derived neurons and characterization of those neurons at the molecular and neurophysiological levels.

The data reported in the *PLoS ONE* paper served as preliminary data for a multi-PI NIH U01 on multiscale network analysis of genetic linkage and gene expression data (with five-year total costs of $10 million). Principal investigators included Gandy and Noggle, as well as Eric Schadt, Ph.D., professor and chairman of the Department of Genetics and Genomics and the Icahn Institute of Multiscale Biology at ISMMS. In addition to Gandy, Noggle, and Schadt, other investigators at ISMMS were involved, along with several at Thomas Jefferson University in Philadelphia and the National Center for Geriatrics and Gerontology in Aichi, Japan.

Cells for the PS1 study were acquired from the Coriell Cell Repository, and those Coriell cells were determined to have karyotypic abnormalities that could confound results. In collaboration with Rudy Tanzi, chairman of Cure Alzheimer's Fund's Research Consortium, three siblings (two affected, one nonaffected) from a family bearing a mutation in PS2 were identified. In early 2014, these subjects traveled to New York and underwent skin biopsies processed by Noggle at NYSCF, thereby providing our first mutant PS2 fibroblasts for preparation of iPSCs and hopefully providing starting cells without the karyotypic changes seen in the Coriell cells. Presumably those changes are due to prolonged storage and/or multiple passages and/or other technical issues.

Noggle and Gandy also used data from the *PLoS ONE* paper as well as unpublished data to prepare an R01 proposal in response to a National Institute on Aging Request for Application (RFA) in Fall 2013. That proposal involves collaboration with the labs of Tessier-Lavigne and Sangram S. Sisodia, Ph.D., (University of Chicago) on projects that will enable genomic editing of typical and atypical pathogenic mutations and/or risk genes reported to associate with AD (e.g., PRGRN, MAPT). Effects of mutations on neurogenesis will be the focus of the collaboration with Sisodia. Other mutations and polymorphisms also will be studied in collaboration with Tanzi to characterize the effects of the new cadre of risk factors discovered in his lab by whole genome/exome sequencing and other strategies. >

The following SCC members did not directly receive funding from Cure Alzheimer's Fund in 2013:

Kevin Eggan, Ph.D.
Harvard Medical School/
Howard Hughes
Medical Institute
Funded through the
Harvard Stem Cell Institute

Marc Tessier-Lavigne, Ph.D.
The Rockefeller University
Funded in 2012

Cholinergic neurons that connect the basal forebrain to the cortex and hippocampus are highly susceptible to amyloid "poisoning" and therefore become dysfunctional early in AD and underlie the typical short-term memory problems. Eggan's lab is focusing on the creation of this specific subtype of neurons in order to enable improved and patient-specific studies of Abeta toxicity in neuronal and mixed cultures derived from iPSCs.

Tessier-Lavigne's lab focuses on perfecting the genomic editing strategies and on the differentiation of neurons representative of those that occur in specific brain regions susceptible to pathology in AD. The MAPT and PRGRN mutations mentioned above will dovetail well with the Tessier-Lavigne and Eggan labs' work since they are studying how these mutations cause a different disease, known as the amyotrophic lateral sclerosis—frontotemporal dementia (ALS-FTD) spectrum disorders. MAPT, PRGRN and other genes carry mutations that cause neurofibrillary tangle formation, dementia, and changes in personality and language. The structure of this collaboration is ideal, since Gandy and Noggle will be studying how mutations in these genes cause AD, while Tessier-Lavigne and Eggan are studying how different mutations in these identical genes cause an entirely distinct dementia. >

Sam Gandy (center) and his reserch team.

Stem Cell Consortium (Continued)

In 2013, Abeliovich and colleagues claimed to have linked APOE isotype to expression of APP-sorting molecules by employing iPSC-derived neurons and *E. coli*-derived apoE. This recombinant apoE is not lapidated or packaged into discs as it is in astrocytes. Therefore, the claims that APOE isotype effect is due to regulation of transcription of APP-sorting protein must be tested using physiologically relevant apoE isoforms. Noggle and Gandy, in collaboration with David Michael Holtzman, M.D. (Washington University, St. Louis) will employ stably overexpressing primary astrocyte cultures as a source of CNS-relevant apoE isotypes. Holtzman has established that partially purified lipoprotein particles from these cultures are structurally identical to those found in brain. In addition, the experiments will be repeated employing mixed neuron-astrocyte cultures using *APOE ε3/ε3* and *ε4/ε4* fibroblasts from siblings.

Along this same line, Kim has made extraordinary progress in developing culture conditions that will enable the production of amyloid deposits and neurofibrillary tangles in human fetal/iPSC-derived neurons in tissue culture. A manuscript describing this system recently has been submitted for publication.

In a logical next step up from the use of iPSC-derived neurons in culture for studying neurophysiology and molecular pathology, Ben-Hur and colleagues are transplanting those iPSC-neurons into the brains of mice, after which researchers characterize how the brain parenchyma alters the molecular properties of the cells, whether and how they develop pathological structures, and how brain neurotrophic and/or neuroinflammatory mediators modulate the viability of the implants and/or the inflammatory response to the cells and/or to the pathology that the cells induce. Noggle and Gandy will begin collaborating with Ben-Hur in the second year, supplying iPSC-derived neurons from the PS2 mutant carriers and unaffected sibling for Ben-Hur's transplant experiments.

In a related spinoff project, Tanzi will be studying the actions of various novel gamma secretase modulators (GSMs) on the speciation of Abeta by the neurons from these PS2 mutation-carrying subjects. An eventual goal is the use of iPSC technology to predict which of the 200+ PS1/PS2 mutations respond best to which GSMs.

In summary, the Cure Alzheimer's Fund Stem Cell Consortium, within the space of one year, has developed highly productive collaborations wherein each lab employs its particular expertise to build one of the various technological way stations that mark the "bench-to-bedside" pathway, culminating in a trajectory that, within a short time, should yield information relevant to personalized pharmacogenomics recommendations in subjects with the most common deterministic cause of hereditary AD.

Abeta Oligomers and the Pathogenic Spread of Tau Aggregation: Implications for Alzheimer's Disease Mechanism and Treatment

A T

February 2013

$126,000

Dominic Walsh, Ph.D.

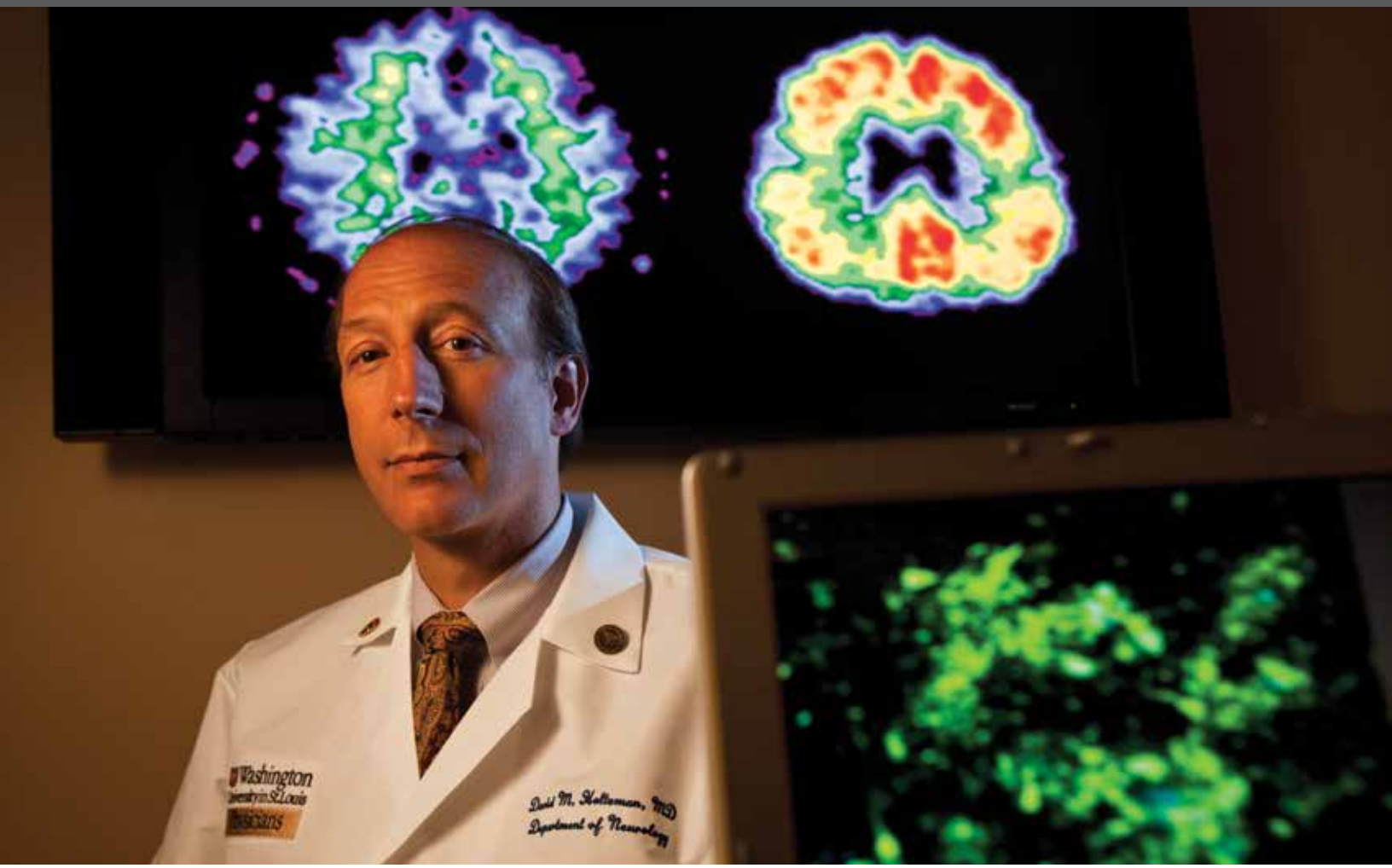
Dennis Selkoe, M.D.

Brigham and Women's Hospital

The goal of this project is to conduct a series of experiments designed to elucidate the role of Abeta and exosomes (vesicles involved in "cell-to-cell signaling") in the transfer of tau clumps from nerve cell to nerve cell.

Two proteins are known to be critically involved in Alzheimer's disease: Abeta and tau. Both are prone to "self-associate," such that in the Alzheimer's brain clumps of Abeta, known as amyloid plaques, are found in the spaces between nerve cells, and clumps of tau, known as neurofibrillary tangles, are found within nerve cells. Until recently it was assumed that Abeta had to form plaques to be toxic; however, it now is clear that smaller, mobile clumps of Abeta (referred to as oligomers) also are damaging. When Dr. Walsh's lab isolated an oligomer from a human brain composed of just two Abeta molecules (referred to as Abeta dimer) and injected it into rats, it caused amnesia. Studies also show that lowering tau levels can protect nerve cells against the toxic effects of Abeta oligomers. These data indicate that Abeta oligomers cause changes in tau that harm brain cells. In parallel, evidence has emerged that clumps of tau can be passed from one nerve cell to another. This process may explain why neurofibrillary tangles appear to spread through the brain as the disease progresses.

Understanding how tau pathology is "transmitted" and whether Abeta is involved should identify novel targets for therapeutic intervention. For instance, if Abeta is found to cause the release of tau via small membranous vesicles known as exosomes, it should be possible to prevent either the release of tau-containing exosomes or their uptake by unaffected recipient cells. If this is possible, drugs designed to prevent the spread of tau pathology should halt further cognitive deterioration. Accordingly, this project will include a series of experiments designed to elucidate the role of Abeta and exosomes in the transfer of tau clumps from nerve cell to nerve cell.



Sleep and Tauopathies: Effect of an Anti-Tau Antibody

October 2013

$100,000

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

In neurodegenerative diseases known as the tauopathies (e.g. progressive supranuclear palsy, Alzheimer's disease), there is progressive degeneration of specific brain regions that account for the symptoms and signs of each disease. Accumulation of aggregated forms of the protein tau in structures known as neurofibrillary tangles and dystrophic neurites in these brain regions correlates well with functional decline in cognition, motor and other functions. Cell-to-cell transmission of tau aggregates leading to brain dysfunction is one hypothesis that may account for the spread of pathology and progressive brain dysfunction. Our recent data, now in press, showing the effectiveness of certain anti-tau antibodies as a potential therapy, supports this hypothesis. One difficulty in assessing the effects of therapies for neurodegeneration in animals is the lack of a strong, quantifiable, physiologically relevant phenotype. Here, we seek to further characterize preliminary findings that a mouse model of tauopathy (P301S Tau Tg mice) develops both decreased NREM sleep as well as a marked decline in delta power during non-REM (NREM) sleep. In addition, we will determine whether an anti-tau antibody, HJ8.5, which we have found to strongly decrease tau pathology, will prevent this sleep phenotype when administered peripherally.

Vascular Regenerative Therapy for Alzheimer's Disease

November 2013

$100,000

Guojun Bu, Ph.D.
Mayo Clinic Jacksonville

Abeta peptides deposited in the brain parenchyma as senile plaques and in the cerebrovasculature as cerebral amyloid angiopathy (CAA) are both hallmarks of AD pathology. Epidemiologically, cerebrovascular damages caused by diabetes mellitus or stroke increase the risk for AD. Cerebral hypoperfusion precedes cognitive decline and neurodegeneration in AD. Our recent work also has demonstrated that cerebrovasculature plays critical roles in Abeta clearance. Therefore, we aim to develop novel regenerative therapy for AD by restoring cerebral vasculature function and neural integrity through transplantation of induced pluripotent stem cell (iPSC)-derived specialty cells. Specifically, we will co-inject iPSC-derived vascular progenitor cells (VPCs) and neural stem cells (NSCs) into mouse brain to promote synergistic effects for regeneration of both cerebral vessels and neurons, as neurovascular interactions play critical roles in neurogenesis and angiogenesis. Therefore, the overall goal of this proposed study is to investigate the effects of regenerative therapy through transplantation of iPSC-derived VPCs and neurospheres on Abeta clearance, amyloid pathology and cognitive function in amyloid model mice. Our innovative approach could lead to the development of novel therapeutic methods to treat AD.

Discovery of Alzheimer's Disease Blood Biomarkers Using Phage Display Technology

December 2013

$100,000

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

Absence of biomarkers has posed a formidable challenge in the development of effective treatment for Alzheimer's disease (AD). Blood-based biomarkers could offer advantages that allow for early AD diagnosis and are critical in monitoring efficacy in clinical studies. Proposed studies aim to identify a set of novel blood biomarkers and examine their potential application as diagnostic agents. Phage display is a powerful approach to engineer peptides or proteins for binding to targets of interest. Therefore, we will apply phage display technology to identify peptides that selectively interact with molecules in AD blood samples, not in the age-matched controls. In Aim 1, we will identify potential biomarkers by screening two libraries with a diversity of approximately 2 billion peptides against AD and control blood samples. To overcome the anticipated kinetic limitations with monovalent peptides, we will polymerize them by conjugation to dendrimers combined with functional moieties, including fluorescent dyes for validation studies in Aim 2. These studies will identify a set of peptides that potentially can be used as diagnostic agents for AD. Furthermore, the proposed research is highly transformative and can be widely applied for biomarker studies in other human disorders. Overall, these proposed studies address a critical unmet medical need in AD by providing large sets of new biomarkers for rapid and accurate noninvasive diagnosis of AD using innovative approaches.

Effects of Inhibitors of Monoacylglycerol Lipase on Behavior and Synaptic Plasticity of Ts65Dn Mice, a Genetic Model of Down Syndrome

A

May 2013

$100,000

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

Alzheimer's disease (AD) is caused by a complex interplay between genetic, epigenetic and environmental factors. Mutations in three genes, amyloid precursor protein (APP), presenilin 1 and 2 account for early-onset autosomal dominant AD (Bertram and Tanzi, 2012). People with Down syndrome (DS) carry an extra copy of chromosome 21, which contains a copy of the APP gene. As a result, by the fourth decade, all people with DS exhibit the AD-type neuropathology and most go on to show dementia by age 60. Thus, DS can be regarded as a valid and robust model of AD. We refer to the occurrence of AD in people with DS by using the term DS/AD. Mouse genetic models of DS carry an extra copy of genes homologous to those on human chromosome 21. One of the best current genetic models of DS, Ts65Dn mice, exhibit abnormalities in brain structure and cognition similar to those observed in DS people, including the degeneration of specific neuronal populations, an effect shown to be caused by increased gene dose for APP.

Monoacylglycerol lipase (MAGL) is an enzyme that belongs to the serine hydrolase family. It was shown that a selective antagonist of MAGL, JZL184, restored to normal the levels of pro-inflammatory eicosanoids and inflammatory cytokines in a mouse model of AD (Piro et al., 2012); inactivation of MAGL also robustly suppressed production and accumulation of β-amyloid (Abeta), a peptide product of APP, and improved synaptic plasticity and memory (Chen et al., 2012).

Our therapeutic hypothesis: Inhibiting MAGL will reduce AD-related neuropathology, restoring to normal measures of inflammation, APP processing, synaptic plasticity and cognition.

The goal of this proposal is to validate monoacylglycerol lipase as a therapeutic target for ameliorating AD-type neuropathology in DS, and by extension AD, thereby providing a novel approach to the treatment of these disorders.

Elucidation of the Mechanism of Action of Gamma Secretase Modulators

A

June 2013

$150,000

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

This project focuses on ultimately defining the structure of a soluble gamma-secretase modulator (SGSM)-bound gamma-secretase enzyme complex at high resolution. Defining the structure of this complex will provide critical information toward elucidating the mechanism of action of this promising series of therapeutic molecules known as SGSMs. This structural information will enable molecular dynamics simulations and can be used to identify critical sites of interaction between the SGSMs and their molecular target, which we have shown to involve the catalytic subunit of the gamma-secretase enzymatic complex.

These studies should enable a critical understanding of the mechanism of how these molecules selectively attenuate only the most pathogenic of the Abeta peptides, e.g., $Abeta_{42}$, and could pave the way for identifying perhaps even more potent and more selective therapeutic agents for the treatment of Alzheimer's disease.

Normalizing Abeta Synaptic Depression with Drugs Targeting PICK1

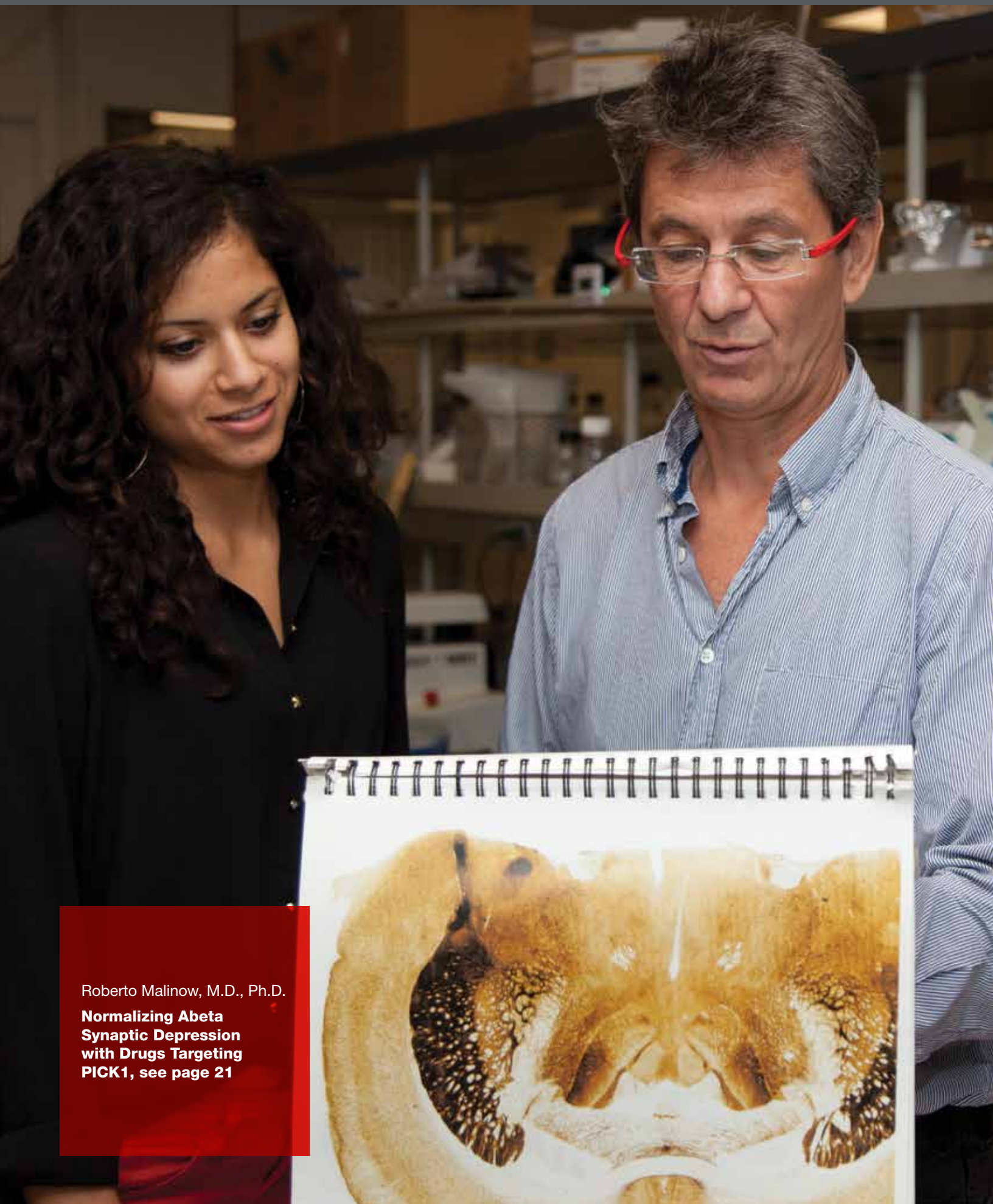
A T

February 2013

$100,000

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

There is general agreement that Abeta is a likely causative agent in the development of Alzheimer's disease. There is growing evidence that early in the disease, an important target of Abeta is the synapse, the site of communication between neurons. We have found that exposure of synapses to Abeta causes their weakening. In this proposal we will examine the role played by PICK1, a protein that associates with synaptic receptors and participates in the weakening of synapses by Abeta. We will test drugs that inhibit the interaction between synaptic receptors and PICK1; such drugs should act to normalize synaptic strength in the presence of elevated Abeta. These drugs may be lead compounds in the search for drugs to treat Alzheimer's disease.



Roberto Malinow, M.D., Ph.D.

Normalizing Abeta Synaptic Depression with Drugs Targeting PICK1, **see page 21**

OUR SCIENTIFIC LEADERSHIP

Research Consortium

The volunteer members of the Research Consortium develop and update a "roadmap for research" for the most effective and efficient route to slowing, stopping and/or reversing Alzheimer's disease. Members contribute their own research projects consistent with that roadmap, as well as recruit others whose work will hasten development of effective therapies for and prevention of Alzheimer's disease.

Rudolph Tanzi, Ph.D., RC Chairman; Harvard Medical School/Mass. General Hospital

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

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

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

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

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

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

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

Sangram Sisodia, Ph.D., University of Chicago

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

Scientific Advisory Board

Members of the Scientific Advisory Board (SAB) are invited independently of the Research Consortium to provide advice and counsel to Cure Alzheimer's Fund regarding the overall scientific soundness of the roadmap and to review individual grant proposals for consistency with the roadmap and with the objectives of Cure Alzheimer's Fund.

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

Dennis Choi, M.D., Ph.D., Stony Brook University

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

Paul Greengard, Ph.D., The Rockefeller University

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

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

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

Thomas Südhof, M.D., Stanford University

Marc Tessier-Lavigne, Ph.D., The Rockefeller University

Full bios available online at www.curealz.org/about/people.

THE PROBLEM

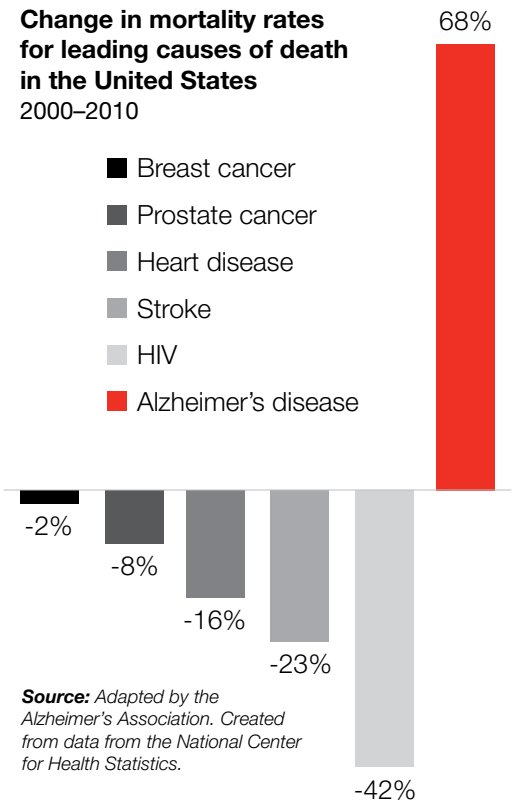
Alzheimer's disease is a progressive, fatal neurological disorder, and it is among the leading causes of death in the United States. While deaths from other major diseases have declined significantly in recent years, deaths from Alzheimer's are on the rise, with a 68 percent jump from 2000 to 2010.

The risk of getting the disease increases dramatically with age. About 10 percent of 75-year-olds have Alzheimer's; some 20 percent of 85-year-olds have it; and about half of everyone older than 85 has it. No one is immune, though certain genetic factors can reduce the risk in some and increase it in others. There is no cure or effective treatment. As of 2012, an estimated 5.4 million Americans had Alzheimer's disease. This includes 5.2 million people ages 65 and older, and some 200,000 patients younger than 65 who have early-onset Alzheimer's. Payments for care in 2012 were estimated to be $200 billion—and more than 15 million Americans provided unpaid care for persons with Alzheimer's. Without a cure, these figures will nearly triple by the year 2050.

The emotional and physical cost always has been devastating to every family dealing with Alzheimer's. Now, with aging populations, the disease also threatens to engulf the health care system of every industrialized nation. Even so, research to cure Alzheimer's is extremely underfunded. Currently, the government spends only $503 million per year on research—far less than what is being spent on care.

Even with adequate funding, curing this disease will not be easy. Alzheimer's pathology starts 15 years or more before symptoms become noticeable, and scientists now think we need to tackle the disease in these pre-symptomatic stages to effectively fight it. Because many factors contribute to the development of the pathology, successful treatment may take the form of a "cocktail" of drugs, which target the disease at different stages of its progression.

Change in mortality rates for leading causes of death in the United States
2000–2010



Source: *Adapted by the Alzheimer's Association. Created from data from the National Center for Health Statistics.*

About **1/2** of everyone older than 85 has Alzheimer's.

An estimated **$200 billion** is being spent on Alzheimer's care per year.

The government spends only **$503 million** per year on research.

OUR APPROACH

100%
of all funds raised
by Cure Alzheimer's Fund
go directly to research.

For many years, Alzheimer's disease research was completely stifled by a lack of funding. Pharmaceutical companies were too wary of past failures to fund any new drug development. The drug pipeline was coming up dry, and researchers weren't encouraged to think big or bold.

Cure Alzheimer's Fund has helped change that. We are a 501(c)(3) nonprofit organization founded in 2004 by three families frustrated by the slow pace of research. Leveraging their experience in venture capital and corporate start-ups, our founders (Henry McCance, Phyllis Rappaport and Jacqui and Jeff Morby) came together to build a new venture-based Alzheimer's research fund designed to dramatically accelerate research, make bold bets and focus exclusively on finding a cure.

The founders
cover
all
overhead
expenses.

Since its founding, Cure Alzheimer's Fund has contributed more than $22,500,000 for research, and its funded initiatives have been responsible for several key breakthroughs—including a potential treatment recently selected by the National Institutes of Health (NIH) for its elite "Blueprint" drug discovery program. Our Research Consortium is an all-star team of scientists working at premier research institutions across the country, regularly conferring with one another on the progress and impediments in their research and constantly sharing their data.

Cure Alzheimer's Fund supports some of the best scientific minds in the field of Alzheimer's research, and it does so without any financial gain for its founders, donors or researchers. Fully 100 percent of funds raised by Cure Alzheimer's Fund go directly to research—the founders cover all overhead expenses.

Our goal is to stop Alzheimer's disease through early prediction, prevention and effective intervention in those patients who have become symptomatic. Please join us in the quest for a cure.

Cure Alzheimer's Fund
has contributed more than
$22,500,000
for research.

OUR RESEARCH INFLUENCE

Cure Alzheimer's FUND 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 or other funders. This "pump priming" is proving increasingly successful, as more of our early-stage grants are leveraged into more substantial and longer-term funding. Another indicator of success is the number of peer-reviewed papers that Cure Alzheimer's Fund researchers have published, and the number of times those papers have been cited by other investigators.

OUR APPROACH WORKS.

2013 grant recipients include*:

- Roberto Malinow, M.D., Ph.D., Synaptic Depression/PICK1 project, received NIH R01 grant
- Sam Gandy, M.D., Ph.D., Oligomer Consortium project, received two VA MERIT grants
- Sam Gandy, M.D., Ph.D., and Scott Noggle, Ph.D., Stem Cell Consortium project, received NIA R21 grant and NIA U01 grant

*As reported by Cure Alzheimer's Fund-funded researchers.

NOTE: For a full listing of published papers supported by Cure Alzheimer's Fund, see **www.curealz.org**.

OUR PROGRESS, YOUR SUPPORT

Message from the President

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

Dear Friends,

This is a profoundly exciting time for everyone connected to **Cure Alzheimer's Fund**. 2013 was a record year both for research and fundraising, and 2014 should be even more fulfilling. Recent breakthroughs we've made in understanding the genetics and mechanisms of Alzheimer's now have positioned us for a quantum leap in research aimed at stopping it.

The researchers, of course, have the really difficult task. But it's also a welcome challenge to efficiently manage your incoming generosity to support our scientists. Let me give you an overview of our approach to this challenge. We received in 2013 the largest amount of contributions for research in our nine-year history—$7.3 million, which enabled us to distribute about $1 million more to research than in the previous year. That growth in our research funding was made possible by a significant increase in the number of donors.

Specifically, we enjoyed the support of about 3,000 donors in 2012, and 5,500 donors in 2013, an 83% increase in the number of people supporting research through Cure Alzheimer's Fund! And we achieved all this with about a 5% drop in total operating expenses from 2012 to 2013.

The accompanying charts tell the story in the aggregate—marked increases in both contributions and research support over the last two years.

If you look closely at the chart, you'll probably ask yourself one big question: we've raised approximately $38 million, but have distributed only approximately $23 million. *What about the other $15 million?*

The answer has two parts. First, approximately $8 million of that money comes directly from our founders and board members over the last nine years in order to pay all administrative expenses. This represents a tremendous and rare commitment on the part of the founders and board to fund all expenses so that every dollar of every other contributor can go directly to research.

As for the other $7 million, we've come to realize how important it is to assure our researchers that our support will be steadfast, regardless of potential peaks and valleys in our fundraising. So we've established a target for reserves—a "rainy day" fund, if you will. We have also, in our nine years of funding research, skewed toward bigger, longer, more complex projects. (Two examples: Our Whole Genome Sequencing project, which we committed in 2012 to fund for $5.4 million, and our Stem Cell Consortium, which called for $600,000 in two successive years). These types of projects require larger sums of money that must be saved and distributed over a longer time span that our typical one-year grant. So that's exactly what we've done.

On top of all this, we have built an "accelerator" fund for just this moment. With it, we are ready to provide initial funding to jumpstart progress. This will help get us started, but we also will need more support than ever from our donors in order to accomplish the objective we all hold dear—an end to Alzheimer's disease.

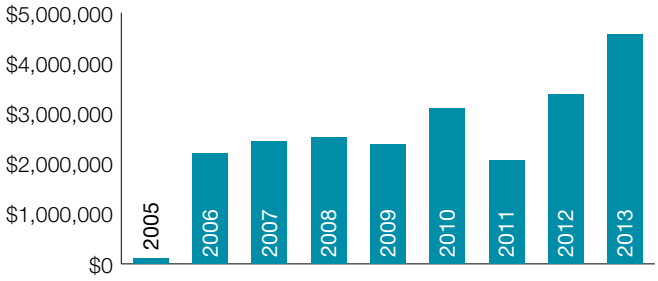
What the charts and these numbers do not reveal is the level of commitment, passion, impatience and yes, even anger, among the growing number of people supporting this effort. To those who put their energies into runs, walks, swims, climbs, tournaments and other events to help defeat the disease, we and the researchers we all support together are deeply grateful.

It is rare to have this many donors stay with a cause at such a high level for so long. And it is also affirming to welcome those new donors supporting Cure Alzheimer's Fund at all levels.This is gratifying, humbling and energizing for all of us who work on this every day. On behalf of the more than 18 researchers who benefited from the help of all those who contributed this year, I thank you. And for those of you who are new to Cure Alzheimer's Fund, we welcome you to this great cause.

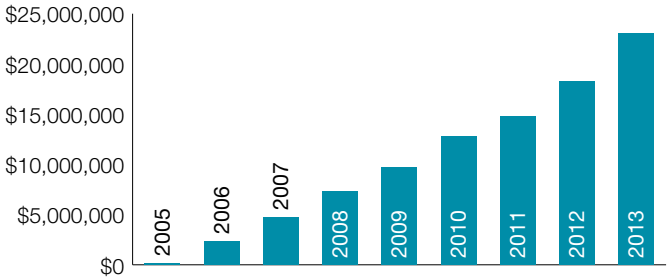
Sincerely,

Tim Armour
President and CEO

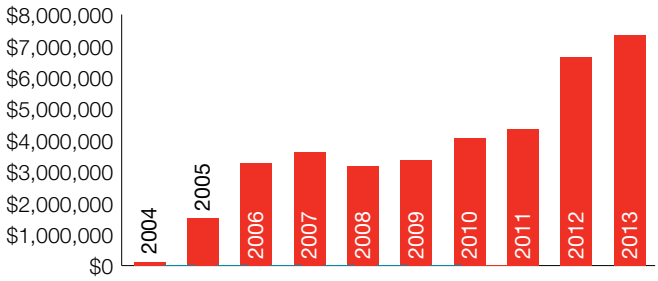
Annual Research Grants



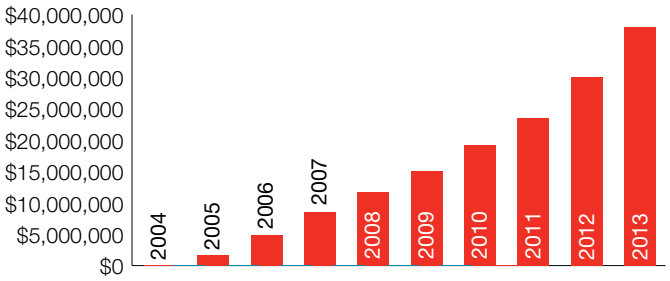
Cumulative Grant Totals



Annual Donation Trends



Cumulative Donation Totals



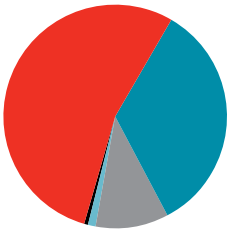
Funding Our Vital Research

- Founders pay for all Cure Alzheimer's Fund (CAF) expenses as well as contribute to research.
- 100 percent of all other (non-founder) contributions go to research.
- CAF does not support overhead or indirect costs at recipient institutions.
- CAF has no endowment and passes all funds raised directly to researchers.
- CAF keeps all funds in cash equivalents; there is no endowment or investment fund, as the objective is to move money from donors to research as quickly as possible.
- CAF funds only projects approved by its Scientific Advisory Board. While proposal approval is as streamlined as possible to facilitate a focus on results rather than process, there is a high premium on the integrity of the science.
- CAF has its IRS Form 990 and audited financial statements online at its website, www.curealz.org. CAF has a history of "clean" audits.
- For the second consecutive year, Cure Alzheimer's Fund has been awarded the highest rating for sound fiscal management and commitment to accountability and transparency by Charity Navigator, the country's largest evaluator of charities.

2013 FUNDRAISING

In 2013, Cure Alzheimer's Fund (CAF) received financial support from individuals, corporations and foundations in the amount of $7,334,757 from 5,500 donors in cash and in-kind revenues.

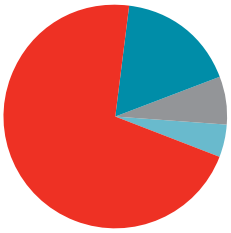
Source of Funds	$	%
Individuals	3,967,850	54.1%
Founders/Board	2,484,413	33.9%
Foundations/ Trusts/Bequests	771,784	10.5%
Corporations	81,268	1.1%
Donated Goods and Services	29,442	0.4%
Government	—	0%
Total	**$7,334,757**	**100%**



Source of Funds

Use of Funds	$	%
Distribution to Research (grants)	4,576,000	71.3%
Programs	1,114,144	17.4%
Management and General	443,283	6.9%
Fundraising	285,049	4.4%
Total Expenses	**$6,418,476**	**100%**

Source: IRS Form 2013 990, now posted on www.curealz.org.



Use of Funds

2013 FINANCIALS (Year ended Dec. 31, 2013)

Statement of Financial Position

ASSETS	
Cash and cash equivalents	$6,268,431
Restricted cash, documentary project funds (temporarily restricted)	191,728
Contributions receivable and undeposited funds	1,062,283
Pledges receivable (temporarily restricted)	1,221,725
Grants receivable (temporarily restricted)	183,333
Deposits – donor-advised funds	8,784
Fixed assets, net	3,226
Other assets	7,990
TOTAL ASSETS	$8,947,500
LIABILITIES AND NET ASSETS	
Liabilities	
Accounts payable and accrued expenses	$110,402
Unexpended authorizations	150,000
Total liabilities	260,402
Net assets	
Unrestricted	7,273,645
Temporarily restricted	
Pledges receivable	1,221,725
Documentary project	191,728
Total temporarily restricted	1,413,453
TOTAL NET ASSETS	8,687,098
TOTAL LIABILITIES AND NET ASSETS	$8,947,500

Statement of Activities

REVENUE AND OTHER SUPPORT	
Contributions	$6,138,347
Net assets released from restrictions (pledges)	$1,238,479
Donated services	29,442
Investment income	296
Realized gain (loss) on sale of stocks	(16,229)
Unrealized gain (loss) on donor-advised funds	(521)
Other income	10,000
Net assets released from restrictions (documentary project)	250,135
TOTAL REVENUE AND OTHER SUPPORT	7,649,949
EXPENDITURES	
Program expenses	
Grants distributed	$4,576,000
Documentary program expenses	250,133
Other program expenses	864,011
Total program expenses	5,690,144
Management and general	443,283
Fundraising	285,049
TOTAL EXPENDITURES	6,418,476
INCREASE IN UNRESTRICTED NET ASSETS	1,231,473
TEMPORARILY RESTRICTED NET ASSETS	
Documentary project contributions	250,000
Net assets released from restrictions	(1,488,614)
INCREASE IN TEMPORARILY RESTRICTED NET ASSETS	(1,238,614)
CHANGES IN NET ASSETS	(7,141)
NET ASSETS, beginning of year	8,694,239
NET ASSETS, end of year	$8,687,098

From the 2013 audited statements which, along with IRS Form 990, is available online at www.curealz.org.

"A close friend of mine lost her mother recently after a lengthy battle with Alzheimer's. Seeing the effects of this horrible disease…has really shown me just how important it is to find a cure."

—Ann Bulson (Boston Marathon)

curealz.org/heroes/ann-bulson

"I ran for my grandfather who passed away from Alzheimer's, and how much I miss him. Throughout my training, many people talked to me about their family members with Alzheimer's and I realized that this disease affects so many lives. I ran to find a cure."

—Kim Chan (Orange County Half-Marathon)

Kim Chan with her medal from the race.

curealz.org/events/2013/running-reason

A SPECIAL THANKS TO OUR HEROES

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, swimming events, 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 $280,000 in 2013!

Pictured left to right: Brent McDonald, Christie McDonald, Steve Harvey, Lorie Brinson (Christie's sister), Tim Goodpaster (father of Christie and Lorie), and Karmen Fink (cousin of Christie and Lorie).

curealz.org/heroes/mcdonald-family

L to R, Barbara Geiger and Carolyn Mastrangelo, co-founders of Running 4 Answers.

curealz.org/events/2012/field-event-running-answers-4th-annual-road-race

Josh, left, and Jake Akman

curealz.org/heroes/jake-and-josh-akman

"We definitely plan to make this [the Dick Hollander Open™] an annual event. There's no stopping this train as long as Alzheimer's has no cure."

—Josh Akman (Dick Hollander Open™)

L to R: Rudy Tanzi, Ph.D., Bob DiFilippo, Bobby Zerwick, Jeff and Jacqui Morby, Mike Curren, Cathy Ingham, John Col and Pam DiFilippo.

Alzhernative Sounds, L to R: Ki Oh, Andrea Huynh, Nathan Huynh, Alex Vu and Brian Stump

curealz.org/events/2013/alzheimers-benefit-concert

"The tournament is my favorite day of the year. My entire family, friends and my parents' friends join us each year and a whole lotta love is shared."

—Gregg Johnson (David K. Johnson Golf Tournament)

curealz.org/heroes/david-johnson-foundation

Second Graders Rally for a Cause (Nate, left, and Kyle)

curealz.org/events/2013/second-graders-rally-cause

Mike Napoli and friends

curealz.org/heroes/mike-napoli

"I've learned firsthand that Alzheimer's is not just an 'old person's' disease. Today, [my wife] Marcia has significant speech impairment. She can no longer drive, cook or do the things she used to do as a mother to our kids. There is no cure for Alzheimer's, no way to prevent it and no way to slow it down. Finding a cure is a must."

—Mike Stanford (Branchburg Race Against Alzheimer's, organized by Mike Napoli)

Stephanie and her mom

curealz.org/heroes/stephanie-hansen

Diana Fiske and Hay Harbor participants

curealz.org/events/2013/12/7th-annual-hay-harbor-tennis-tournament

Kelly Hulfachor

curealz.org/heroes/kelly-hulfachor

Greg Wellman Jr. and his grandmother

curealz.org/heroes/greg-wellman

Charlie Collier (right) with Cure Alzheimer's Fund founders Phyllis Rappaport (center) and Henry McCance (left).

curealz.org/events/2013/evening-charles-w-collier-ups-and-downs-living-alzheimers-disease

Nelson Everts

curealz.org/heroes/nelson-everts

Sam Gandy presenting his research.

OUR 2013 EVENTS

Cure Alzheimer's Fund offers free educational opportunities to the public with an annual fall symposium and quarterly Alzstream™ webinars. This year, we also hosted screenings of films about Alzheimer's and its effect on the lives of patients and caregivers. Banker White's "The Genius of Marian" and David Shenk's "Living With Alzheimer's" were shown in conjunction with the symposium.

Our 2013 symposium, held at the Revere Hotel in Boston, featured presentations by Drs. Rudy Tanzi and Sam Gandy. The event was viewed by a record crowd—nearly 200 attended in person and another 500 watched a live video stream on our website.

L to R, David Shenk, Devon Angelini and Ed White discuss "The Genius of Marian."

Rudy Tanzi

Sam Gandy

"As a physician who treats many patients with Alzheimer's disease in my medical practice, it was exciting to attend the Cure Alzheimer's Fund annual symposium to hear about the latest advances in research from the doctors and scientists who are leading the way in finding a cure for this condition in our lifetime. I am most encouraged to know that such highly committed individuals, with the support and involvement of the fund, are accelerating the pace of innovation and discovery in order to cure this disease that profoundly affects millions of families."

—Rob Fields M.D., FACP

Symposium guests watching "Living with Alzheimer's."

WEBINAR

Find out more about

Alzheimer's Research: Public-Private Partnership

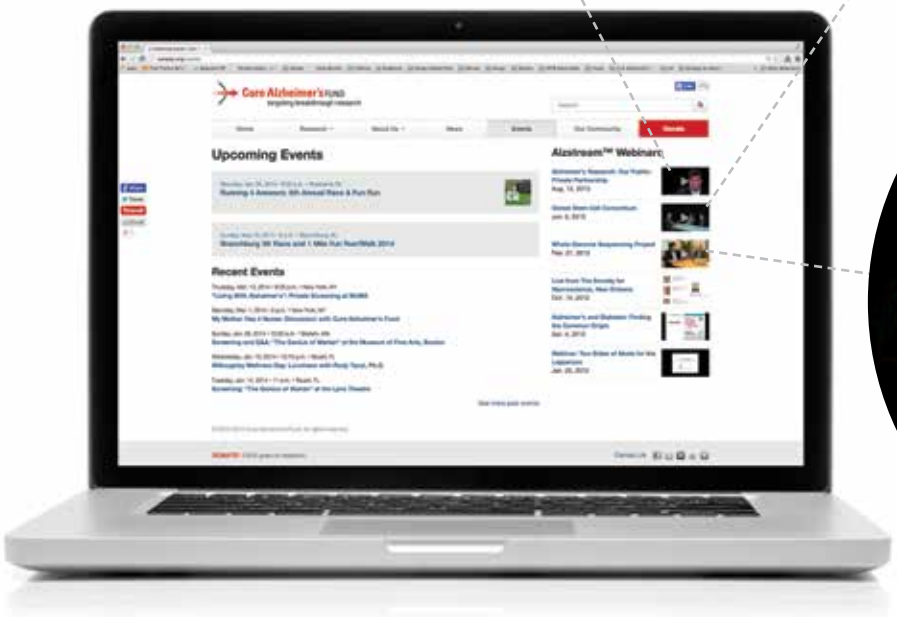
SPEAKERS:
David Shenk
Rudy Tanzi, Ph.D.
Tim Armour

WEBINAR

Find Out More About Cure Alzheimer's Fund's

Stem Cell Consortium

SPEAKERS:
David Shenk
Sam Gandy, M.D., Ph.D.
Scott Noggle, Ph.D.



WEBINAR

Join us for a discussion about the Cure Alzheimer's Fund

Whole Genome Sequencing Project

and its expected impact on Alzheimer's disease.

MODERATORS:
David Shenk
Rudy Tanzi, Ph.D.

ALZHEIMER'S/ DEMENTIA

ROTARIAN ACTION GROUP

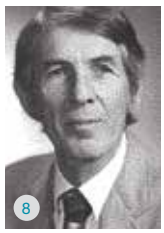
Collaboration with Rotary

In June, the Board of Directors of Rotary International recognized the Alzheimer's/ Dementia Rotarian Action Group, one of a handful of such philanthropic groups within the organization. This was the culmination of a three-year process initiated by Cure Alzheimer's Fund co-founder Jeff Morby, who is a Rotarian in the Martha's Vineyard Rotary Club. This effort included trips to Bangkok, Thailand, and Lisbon, Portugal, where Jeff and Dr. Rudy Tanzi presented to thousands of Rotarians. Their message was very well received: It is urgent that we accelerate the pursuit of curative treatments for this disease. Cure Alzheimer's Fund is honored to be collaborating with such a prestigious organization as Rotary International. This alliance with one of the world's largest and most successful humanitarian organizations holds great promise for the future, and was one of the most rewarding partnerships of our 2013 year.








IN HONOR AND IN MEMORY

Cure Alzheimer's Fund received gifts in honor or in memory of the following in 2013:

Abbas, Kharia
Abbate, Frances
Abdulaziz, Sam
Abramovitch, Rudolph
Ackell, Ed
Adrien, Kristen
Ahern, Margaret
Albini, Caterina
Alexander, Milton 8
Allan, Joyce
Allcroft, Stephen
Althoff, Eunice
Amend, Susan Epps
Amerault, Robert
Anastasion, Christine
Anderson, Hugh and Manilla
Anderson, Jane
Anderson, Marianna 9
Anderson, Mona
Angerstein, Evelyn
Ankenbrand, Eleanor
Ankney, Vernon 1
Apollo, Mary Ellen
Armbruster, Rose
Armentrout, Josh
Armstrong, Anna
Arthur, Rodney
Asselin, Gerard
Ault, Ada 10
Austin, Dorothy
Avant, Lois Slate
Axelrod, Bernard
Ayers, Thomas
Azhederian, Lazarus
Backfisch, Cecilia
Bailey, Lisa
Baker, Sandie
Baker, Tommie Miller
Bakeris, Sadie
Balch, Jack
Baldassari, Marjorie
Ball, Carolyn 11
Ballard, Jean
Ballast, Marjory 2
Barnes, John
Baron, Lois
Barr, Dorothy
Barrett, Mary Anne
Barsam, Margaret
Barstad, Ruth
Bass, Ann
Bauer, Erline
Bauer, Sueann
Baumstark, Rose
Beall, Dixie
Bear, Sylvia
Beard, Bob 12
Beaton, John
Beauregard, John
Beban, Bonnie
Becker, Agnes
Becker, Edith
Becker, Gladys
Becker, Mary
Becker, Mary Agness Collins
Behrens, Alan
Bekker, Dina
Bendix, Lucille
Benjamin, Charles
Bennett, Dave and Molly
Bennett, Mary Alice
Bennett, William
Bensimon, Albert Leon
Berger, Rose
Bergquist, Robert
Bergstrom, Betty
Berkowitz, Selma
Berman, Jack
Berman, Paul
Bert, Elizabeth C.
Betancourt, Ernesto
Betz, Carol
Betz, Janet and Denny
Beyschau, Mabel
Bidwell, Theodore
Billy, Marge
Binder, Evelyn
Binder, Lester "Skip"
Bixler, Bruce
Black, Edy
Blackwell, John
Blake, Parle
Blatnik, Steve
Bleaner, Lee
Blevins, Lida
Blevins, Norma
Blinn, Susanne
Blount, Jan
Boatright, Kenneth
Boatright, Ronald
Bohm, Frank
Boles, Rini
Bonfitto, Lucy
Bono, Blanche
Booth, Brian
Bordewieck, Robert
Borgwardt, Jack
Borwick, Lilian
Bosdell, Grace 13
Bosler, Byron
Boucher, Hazel
Boulanger, Joyce
Boullie, Marjorie Ann
Bovee, Ruth
Bowles, Sylvia
Braham Jr., W. Walter
Brandstatter, Joyce
Branovic, Margaret
Brassard, Lucille
Brauchler, Philip
Breitbard, Robert
Bremner, Gillian
Brennan, Kay 14
Breuer, Marie
Brickle, Francis
Broberg, Jolene
Brockmann, Linda
Brodsky, Melvin
Brolick, Bianca
Brooks, Bill
Brooks, Evie
Brown, Bethel
Brown, Ed and Diane
Brown, Howard
Brown, Richard
Brown, Robert
Brunetti, Tom
Bryan, Virginia
Bryett, Sydney
Buben, Annette
Bucklin, Joan
Bujold, Hazel
Burgesen, Maureen
Burke, Tommie Gene
Burman, Eleanor
Busalacchi, Betty Jayne
Bushnell, Jean
Buskirk, Eleanor Van
Cagle, Lebron
Cahill, Betty
Calandra, Mildred
Calhoun, Buford
Campanella, Roslyn
Campanelli, Nicholas
Campbell, Ina Davis
Campbell, Mary
Campbell, Mary Ellen
Campbell, Urcil
Canfield, Patrick
Canning, Harry
Carlo, John
Carlson, Richard
Carozzoni, Marino
Carroll, Harlean
Carroll, Velva
Caruso, Josephine
Carye, Marge
Casselberry, Thelda
Celette, Ed
Celette, Mary
Cerwonka, Kass
Cetel, Claire
Ch, Kwang-wen
Chalfant, Theresa
Chasin, Carol
Chatham, Anne Stanley
Chatman, Ray
Chatten, Ruth
Chieco, Mary
Childs, Madeline
Chiles, Steve
Chiu, Su
Christian, Dorothy
Chupp, Maury 3
Churchill, Linnea
Clark, Hoy Joe
Clark, Leo
Clark, Mike
Clark, Thomas
Clark, Virginia
Classen, Charlotte
Clemons, Barnetta
Cleverdon, Margery G.
Clinton, Agnes
Cochrane, Una
Coen, Opal
Coffield, Miriam
Cohen, Annette
Cohen, Ben
Cohen, Sumner
Coker, Betty
Colburn, Priscilla
Colby, Elouise
Cole, Rachel
Coleman, Bernice
Commiso, Ellen
Conner, Nola
Conway, Bill
Conway, Tim
Coombs, Esther
Coontz Sr., Robert
Cooper, Barbara Ann
Cooper, Yoko
Cortney, Mary
Cosma, Edward
Cott, Bernard
Coveney, Philip
Cowan, Robert
Cox, Emily and James
Cox, Irene
Craigie, Mary
Cramer, Elizabeth
Cramer, Thomas
Crawford, David
Crawford, John
Cristock, Jack
Critchfield, Carolyn
Critendon, Morris
Crockett, Julia
Crowl, Madlyn
Culpepper, Dale
Cupp, Dorece
Curad, Ariello
Currie, Duncan
Daetwyler, Clella
Daniels, Drexel
Dautrich, Pauline
Davidson, Irving
Davis, Shirley
Day, Robin
Dean, Geraldine
Dearing, Ollie
Deebach, Grace
Deekens, Hilda
Defibaugh, Leota
Dello Buono, Emidio
Demarest, George
DeRoma, Norma
Derrickson, Eleanor
Detelich, Fay
Dever, Alice
DeWitt, Henry Birt
Dichio, Pasquale
Dickson, Constance
DiCola, Helen
Diedrich, Shirley
Diggs, Robert
DiMauro, Agatha
DiPuccio, Angelo
DiSabatino, Constance
Dishart, Theresa
Dolby, Sarah
Donahoe, Dan
Donahoe, J. LaVerne
Dorris, Gertrude
Dorwart, Reinhold and Juanita
Doughty, Jean
Douglas, Anita
Downs, Valerie Corky
Doyle, Austin
Drake, Patricia
Dratler, Libby
Dring, Janet
Driscoll, Shirley
Dubina, Philip 15
Dubson, Leo
Duncan, Melba Jane
Dutt, Sudarshan Kumar
Dwight, Murray
Dworkin, Frank
Dwyer, Steve
Earp, Delores
Eason, Douglas
Eason, Elizabeth
Eastman, E.R.
Ecker, Judy
Eckhardt, Catherine 4
Edgar, Timothy
Edson, James
Egan, Gloria Joan
Eggler, Isabel
Eid, Carolyn
Elkington, George 16
Ellenbrand, Bonnie 17
Ellison, Richard
Ellison, William
Ells, Marguerite
Elms, Morris
Elson, David
Emerson, Phoebe DuPont
Engelage, Don
Eppsteiner, Gary
Epstein, Beth
Ertel, Mildred
Esser, Gordon
Etchepare, John
Everdell, Romeyn
Everts, Nelson
Ewald
Faber, Gerald
Fairly, Lowell
Farrell, Mary
Farwagi, Raymond
Faulkner, Dorothy
Fecher, Mary
Fecteau, Helen Mary
Felker, Kristen
Fellner, Shirley Stolk
Ferris, Donna Moehn
Feuer, Joseph
Finan, Dorothy
Fink, Melba E.
Finn, Julie
Firsching, Kathryn
Fisher, Nick
Fishman, Claire
Fister, Susan
Fladmark, Mary
Flaherty, Eileen
Flamini, Alberta
Fleming, William
Floyd, Tom 18
Fohn, Irene
Fok, Yuen Chun
Foley, Joan


10


11


12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14


17


15


16


18

Foley, Paul
Ford, Shirley
Forman, Beverly
Fornes, Lillian
Forsch, Erna
Fort, Bennie
Fox, Betty
Fox, Marian
Frank, Charlotte
Freeman, Ruth 19
Frohman, Marion
Frost, Betty Ellen
Fujii, Shoji Paul
Furlong, Barbara 20
Gadomski, Lillian
Gagnon, Rosalie
Galarneau, Gerald
Gallagher, Diane
Garretson, Louisa
Gartman, Robert
Gasser, Urban
Gavry, Mary
Gegner, Mae
Gelbwachs, Donna
Gentile, Angelo
Gentner, James
Ghio, Albert
Gibbard, Jacqueline
Gibbons, Folsta
Gibbs, Gordon
Gibson, Doreen
Gibson, Elizabeth
Gillespie, Beatrice
Gillings, Dulcie
Gleason, Bud
Glynn, J. Frederick
Goerges, Josef and Agnes
Golas, Annette
Goldshine, Sheila
Goldsmith, Goldie
Golub, Rosaline
Gonsalves, Joseph
Goodman, Ann
Goodpaster, Edward
Goodwin, Florence
Gorman, James
Goulding, Lola
Governale, Joseph
Graham, Amy
Grandmother Balsman
Grandpa Garcia
Grant, Anne
Graves, Sam
Green, Col John
Gregg, Herbert
Griffin, Sally
Grissom, Charles
Gristock, Jack
Gross, Millie
Grove, James
Grow, Sam
Gu, Yu Hwa
Guiltinan, John
Gunders, Elaine
Gupta, Meera
Gustafson, Miriam
Gustafson, Ruth 21
Gutbrod, Geraldine
Gutzmer, Robert
Gyuricsek, Dana
Haag, Agnes
Haas, Helen
Halis, Loraine
Handel, Hannah
Handlos Sr., Robert
Hardin, Martha Lillian
Harmon, Linwood
Harms, Baulah 22
Harned, D.H.
Harriman, Jolene
Harrington, Wilma
Harrison, Donald
Harstedt, Elinor
Hart, Stephen
Harthman, Adele
Harwood, Lois
Hasenour, Jerome
Haskins, Shirley
Hayner, Dennis and Ruth
Haynes, Wesley
Hays, James and Ann
Heal, James
Heard, Janet
Hedin, Julia
Heffernan, Paul
Henaman, Kathie
Hendee, Theresa
Hendrickson, Roy
Henneman, Jeffrey and Peggy
Herndon, Max
Herning, Don
Herring, Ada
Herring, Laura
Hewitt, Patricia
Heyman, Sandy
Hicks, Jeanette
Hinchey, Dean Jones
Hirsch, Lanore
Hirst, Louise
Hiss, Shirley
Hitchcock, Elizabeth 23
Hladek, Olga
Hoagland, Laurie
Hoff, Harry
Hoffman, Arlene
Hoffman, Rose
Holloway, Georgia Ann
Holmes, Carol
Holmes, George and Nancy
Holt, Henry
Holton, Felicia Antonelli
Hood, Kathleen
Hooker, Stuart
Horne, Howard
Hosford, Georgette
Howard, Bill
Howard, Brian
Howard, Clyde
Howell, Jack
Hubbard, Bo
Huberman, Sam
Huffman, Robert
Hunter, Phyllis
Huntley, Barbara
Hurley, Prudence
Hurst, Grace
Huszczo, Virginia
Hutchings, Rendel
Hutchins, Ani
Hutchison, Helena
Ilg, Frances
Imo, Juliet and Cecilia
Ingram, Carolyn 24
Isenberg, Rita
Ivy, Sue
Izen, Elliot
Jackson, Sandy
Jaffe, Eve
Jahoda, Franz
Jakob, Norma
Jamieson, Vivian
Janway, Arta
Jewell, George
Jodrey, Maxine
Johnson, Charlene
Johnson, Ellyn
Johnson, Jack
Johnson, Maxine
Johnson, Peggy J.
Johnson, Raymond
Johnson, Robert
Johnson, Sigfred
Johnson, Vera G.
Jones, Bobby
Jones, Elizabeth
Jones, Mamie
Jones, Ralph
Jordan, Lynvel
Judd, Kathy
Kadlick, Joan
Kanas, Helen
Kanter, Gertrude
Kantro, Henry
Kaplan, Arthur
Kates, Murry
Kauppinen, Marie
Kawamoto, Auntie Ruby
Kearns, Roger
Keenum, June
Keep, Eleanor
Keil, Evelyn
Keller, Violet Mae
Kellogg III, Edward
Kellogg, Ned
Kelly, Dianne
Kemp, Virginia
Kennedy, Bettie
Kennedy, Sue
Kiell, Gail
Kikin, Regina
Kim, Ryan
Kimball, L. Robert
Kimbley, Bill
King, Evelyn
King, Velda
Kipnes, Edith
Kirkland, Jerry Bob
Klein, Catherine Theresa
Klein, Frances
Kluesing, Marie
Kneeland Sr., John
Koerner, Donald
Kohnen, David
Kononchik, Jere
Kornblum, Carol
Korth, Joseph Vargas
Kossler, Ruth
Kotzen, Herbert
Krassner, Marvin
Krede, Margaret Vosburgh
Kreidewiess, Adeline
Krieger, Jerry
Krom, Dorothy
Kronisch, Alice Grossman
Kropp, Janet
Krueger, Ruth
Krueger, Vernon
Kuhn, Betty
Kulosa, Vera
Kurz Jr., Albert
Kurz, Mary 25
Kutnick, Drew
La Mama
Labadini, Ida
Lackey, Dorothy
Lak, W.P.
Lanari, Rick
Lapinski, Julia 26
Lau, Sui Shan Yip
Lawrence, Sandra
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LeGoff, Yves
Lehman, Camille
Lehman, Emma Lou
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Leitzke, Glenn
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Markey, Edward
Martin-Vegue, Howard
Martin, Douglas
Mascarenas, Helen
Mason, Sue

In 2013 **1,118** people were memorialized through gifts in their honor or memory.















Mason, Vera
Matathia, David
Mayer, Paul
Maytum, Claire
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Nolan, Rebecca
Noonan, Margaret
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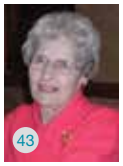

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Turner, Bonnie
Turner, Martha
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Urban, Edith
Urdang, Richard
Vactor, Doris Van
Van Bruggen, Kathryn
Van Vort, Phillip
Vandergriff, Dorothy
Vanderveer, Nancy
Vandeveer, Florence Pat
Vandevoorde, Violet
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Varrell, David
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Venema, Ann
Verna
Vetere, Mary
Vetter, Mary
Vezey, Mary
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Walker, J. Evelyn
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Warnke, Morris
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Weiner, Lucille
Weinstein, Beth
Weisbeker, Rosa
Weiss, Mona
Weitzel, Lloyd
Wellman, Greg
Wessels, Gerald "Chub"
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Wetzel, Jan
Whale, Arthur Dick
Wheeler, Les
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Whitaker, James
Whitcomb, Cheryl
White, Katherine
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Wolf, Meinrad
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Wylie, Jackie
Wyman, Winn
Yanchulis, C. Phillip
Yang, Kuan
Yarmel, Chip
Ye, Peggy
York, Lynn
Young, Arlene
Young, Betty Jean Quick
Young, Jean Marie
Zastrow, Emil
Zelermyer, Andrew
Ziegler, Lee
Ziskind, Richard
Zitelman, Harry
Zola, Sandy
Zoldan, Morris
Zwicker, Lorraine

What better way to pay your respects to your loved one than to give a life-affirming gift in their memory to research into the cause of Alzheimer's disease?

If you would like to designate memorial gifts to come to Cure Alzheimer's Fund, please let us know whom to notify when we receive donations.

Whether gifts are made online, by mail or by phone, we will gratefully acknowledge each donation by notifying the person or family member designated (no gift amount is disclosed).

Photos sent to us are posted on our website under "In Memory."
www.curealz.org/who-we-remember

Each donation will honor your loved one and help sustain our research projects. Thank you for designating charitable contributions to Cure Alzheimer's Fund!

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

Our family would like to extend deep gratitude for all that Cure Alzheimer's does for research into this horrible disease. Watching our mother disappear in front of our eyes was more difficult than words can express. She was a nurturing, caring person....Hopefully in my lifetime a cure or the ability to stop the progression of Alzheimer's is found.

—P. Phelps

Witnessing the control Alzheimer's took over our mother's body and life since she was 59 was very heart wrenching. She was our precious mother who was robbed of her health and time to enjoy life and her family. It is our sincere hope that a cure be found very soon. We miss her dearly.

—Daniela, Marco and Claudio Magarelli

WHY CURE ALZHEIMER'S FUND?

To end Alzheimer's, we believe it's imperative to focus on and fund research that is innovative, collaborative and results-oriented. Our funded work, upholding these values, has made tremendous progress in the search for an Alzheimer's cure. But there is more work to be done.

We invite you to join our ongoing effort to support the most promising, speed-driven, 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)**.

My husband, Wolfgang Roth, age 83, died this past November of heart failure. He was beginning his seventh year of Alzheimer's, and so our family decided not to submit him to heart surgery. We are grateful that he did not have to continue on his quickly escalating slide into dementia.

Wolfgang and I became supporters of the Cure Alzheimer's Fund after reading its stellar review on Charity Navigator two years ago. I have read with great interest the informative annual reports from cover to cover and am proud to be a supporter of an organization that is in the forefront of effective research on AD. Thus, our family has designated the Cure Alzheimer's Fund as a recipient of donations in Wolfgang's memory.

—Mary Beth Roth

Cure Alzheimer's FUND

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.

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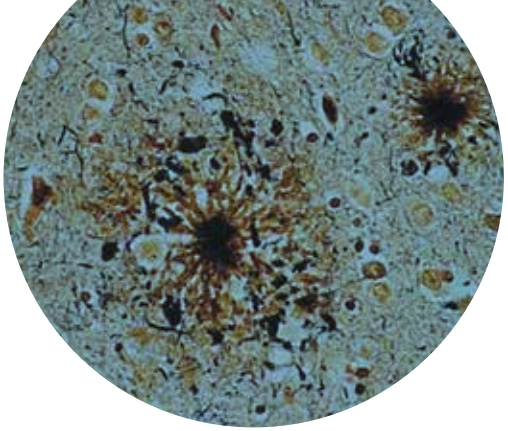

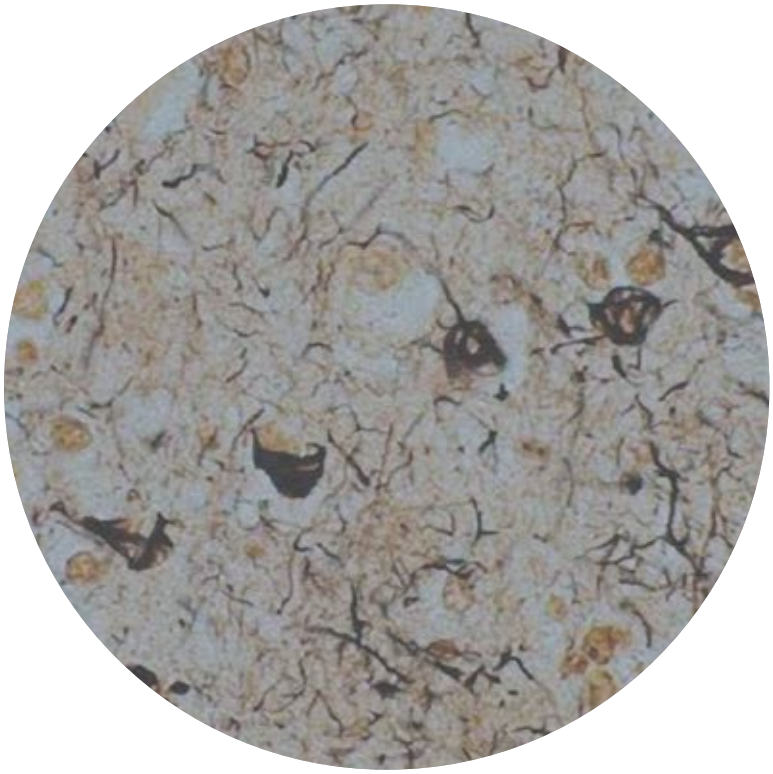

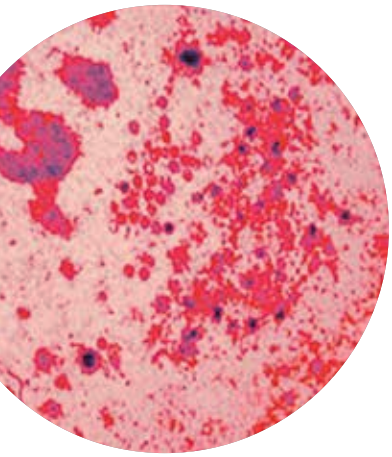

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