Progress Through Research
MISSION
Fund research with the highest probability of preventing, slowing or reversing Alzheimer’s disease.
2010 was an extremely successful year:

- As a result of our research funding, our scientists continue to make tremendous strides in identifying the causes of, and possible preventatives for, Alzheimer’s disease.
- Our distinctive research successes and our focused approach to managing the research process now have been recognized by major institutions and thought leaders throughout the country. We have come to be identified as a “philanthropic model” for others to follow.
- In 2010 we raised a record $4 million in new funds and financed more than $3 million in research, with another $1 million in projects already committed for 2011.

Research Progress

Our scientific accomplishments continue to change the landscape of Alzheimer’s research and carry us all closer to the end of this terrible disease. In particular:

- The Alzheimer’s Genome Project™, funded exclusively by us and completed in its first phase in 2008, continues to provide us with substantial and startling insights. One of these, for example, is that neuronal death and tau formation may be related to a genetic defect in which neural cells, which are not programmed to divide, attempt to do just that and bring about their own deaths. Other insights revolve around the genetic mechanisms by which too much Abeta gets generated or too little Abeta gets cleared from the brain. And there many more. We are now focusing on the top 25 AD genes discovered and systematically piecing together, on a gene-by-gene basis, the “mechanisms of action” that lead to defective genetic action.
- Identification of three potential preventatives (medications) for the disease, which could lead to biotech companies or other entities taking them to the next steps, involving more refinement of the biology and hopefully leading to Stage 1 trials.
- Continued research to unravel the dimensions of the totally new and potentially very significant theory concerning the cause of Alzheimer’s—but now with leveraged funding from the National Institutes of Health. The “anti-microbial hypothesis” explores the concept that Abeta oligomers perform both a positive function—protecting the brain from invading pathogens—and a negative function—damaging neurons when defective genes overproduce the oligomers. This insight ultimately may come to be recognized as the linchpin for multiple approaches to disease prevention.
Through our research we have identified the central causal agents that block neurological transmission leading to lack of memory formation—Abeta oligomers. We have funded considerable research attempting to define which of those Abeta combinations are the most harmful, in an effort to identify the mechanisms by which such oligomers block memory. Those projects, which strike to the heart of the AD issue, continue on with new techniques and new outstanding researchers recently brought into our Research Consortium.

Continued funding of the maintenance and refinement of the online database AlzGene.org, which contains compilations and analyses of all genetic Alzheimer’s disease research worldwide and makes that information freely available to scientists for their use, thereby facilitating the critical sharing of information.

Recognition by Major Thought Leaders:

Through interactions with others, conferences and presentations, including events like TEDMED, the Milken Global conference and a panel at the White House, CAF representatives have made considerable impact in building awareness of Alzheimer’s impact on society and the need for a coordinated nationwide strategy to bring an end to the disease. Below is a small sample of favorable remarks about CAF:

U.S. Rep. Ed Markey: “As co-founder and co-chair since 1999 of the Bipartisan, Bicameral Congressional Task Force on Alzheimer’s Disease and co-author with Representative Chris Smith of the National Alzheimer’s Project Act signed into law on January 4, 2011, I have been in a position to meet with and monitor the activities of a great variety of organizations concerned with Alzheimer’s disease. In my opinion, Cure Alzheimer’s Fund stands out among those for its contributions of bold, innovative and high-risk/high-reward research.”

Bill Sahlman, senior associate dean, Harvard Business School: “CAF is a paragon of best practice. The organization is lean and focused. The founders underwrite all administrative costs so that donations go directly to support research. The founders have deep expertise in applying the venture capital model to allocating funds to great individuals and teams.”

Sean Corcoran, senior reporter, National Public Radio: “For the nation, Alzheimer’s disease is a scourge, a memory-stealing, brain-clogging affliction that will—and I say this without hyperbole—bankrupt the country’s health care system within my lifetime if a preventive is not found…. The reason I decided to spend several weeks reporting on CAF specifically is because I became convinced that this small group of altruistic individuals may succeed. The more I learned, the more it appeared that CAF’s funding scheme, its approach and its founders’ experience just may ‘cure Alzheimer’s’ by finding new treatments and/or preventatives.”

Leveraging Our Record Funding and Insights Through Collaborations With Others:

When we established CAF more than five years ago, one of our stated objectives was to finance high-risk, high-potential research, which in the normal course would not be funded by the NIH and other more conservative funders, with the objective of gaining some truly breakthrough insights, which then could lead to substantial funding or co-funding from or with more traditional funding sources. We are now achieving that objective. Our anti-microbial insight, totally funded by CAF, has resulted in a multimillion, multiyear NIH grant that builds upon our breakthrough findings. Additionally, four of our researchers have been able to obtain NIH grants totaling several million, again, building upon the insights derived from our original funding. Finally, it should be pointed out that our AlzGene data base, freely available to all, will most certainly spawn additional useful research by others, and the Alzheimer’s Genome Project™ (AGP) already is stimulating considerable new productive research.

To conclude, none of the above would have been possible without the generous support of more than 3,500 supporters, including the three founding families, who continue their substantial personal support. Thank you all for the confidence you have placed in us—and your generosity.

Jeffrey L. Morby, Chairman
What Makes Us Unique?

- **We have BIG goals, but one objective.**
  We’re not focused on incremental steps. We are willing to try a 50-yard pass vs. a one-yard plunge at finding a cure.

- **We think like venture capitalists.**
  We bet on the scientists, not on the institutions, and cut out the bureaucracy so they can focus on research.

- **Genes are our compass.**
  We start with genetics because we can’t get to a cure without understanding the cause.

- **Collaboration is key.**
  We find the best people making the most progress on the most important issues and help them work together.

- **Scientifically rooted.**
  While we approach research in a strategic, results-oriented way, we also are committed to and grateful for scientific independence and rigor. We have created a world-class Scientific Advisory Board to attract the best people and their ideas.
Recognized in the News

MassGeneral Magazine
Two Articles—Summer 2010
Cure Alzheimer’s Fund: A Venture Capital Approach to Philanthropy
Unforgettable Breakthroughs in Alzheimer’s

AARP Article Featuring CAF-funded Discovery—September 2010
New Science Sheds Light on the Cause of Alzheimer’s Disease: Researchers now looking in a new direction for a cure

The Wall Street Journal Health Blog—October 2010
TEDMED: Greylock’s Henry McCance Brings VC Approach to Medical Research

CNN.com Featured a Summary of the Tanzi/McCance TEDMED Talk—October 2010
Dr. Rudolph Tanzi of Cure Alzheimer’s Fund laid out an ambitious goal of finding a cure for the illness by the year 2020. Tanzi and venture capitalist Henry McCance, who helped found the fund in 2004, described the partnership that led to the identification of new genes that seem to play a role in Alzheimer’s disease, genes that are potential targets for new drug therapies.

National Public Radio—November 2010
Two-Part Radio Broadcasts:
Venture Philanthropy Part 1: A Business Approach to Curing Alzheimer’s Disease
Venture Philanthropy Part 2: Wanted: A Man on the Moon Project to Cure Alzheimer’s

Online Article: Massachusetts Venture Capitalists Invest in Alzheimer’s Research
In 2010, Cure Alzheimer’s Fund (CAF) received financial support from individuals, corporations, foundations and government in the amount of $4,013,000 from 1,230 donors in cash and in-kind revenues.

### Source of Funds

<table>
<thead>
<tr>
<th>Source of Funds</th>
<th>$</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Founders</td>
<td>1,749,000</td>
<td>44%</td>
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<tr>
<td>Non-Founder Individuals</td>
<td>1,290,000</td>
<td>32%</td>
</tr>
<tr>
<td>Foundations</td>
<td>703,000</td>
<td>17%</td>
</tr>
<tr>
<td>Government</td>
<td>148,000</td>
<td>4%</td>
</tr>
<tr>
<td>Corporations</td>
<td>93,000</td>
<td>2%</td>
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<tr>
<td>In-Kind Donations</td>
<td>30,000</td>
<td>1%</td>
</tr>
<tr>
<td>Bequests</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>$4,013,000</strong></td>
<td><strong>100%</strong></td>
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</table>

### Use of Funds

<table>
<thead>
<tr>
<th>Use of Funds</th>
<th>$</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Distribution to Research (grants)</td>
<td>3,094,000</td>
<td>76%</td>
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<tr>
<td>Grant Support/Programs</td>
<td>579,000</td>
<td>14%</td>
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<tr>
<td>Management and General</td>
<td>226,000</td>
<td>6%</td>
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<tr>
<td>Fundraising</td>
<td>186,000</td>
<td>4%</td>
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<tr>
<td><strong>Total Expenses</strong></td>
<td><strong>$4,085,000</strong></td>
<td><strong>100%</strong></td>
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</table>

Source: IRS Form 2010 990, now posted on [www.curealz.org](http://www.curealz.org).

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- Founders pay for all Cure Alzheimer’s Fund (CAF) expenses as well as contribute to research.
- 100 percent of 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.
- A corollary of the “no endowment” policy is that CAF suffered virtually no loss of assets during the recent financial crisis. Donor money was safe and deployed to research as planned.
- 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 online at its website, [www.curealz.org](http://www.curealz.org); audited financial statements are available upon request. CAF has a history of “clean” audits.
Statement of Financial Position

ASSETS

<table>
<thead>
<tr>
<th>Description</th>
<th>Amount</th>
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</thead>
<tbody>
<tr>
<td>Cash and cash equivalents</td>
<td>$2,998,023</td>
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<tr>
<td>Contributions receivable and undeposited funds</td>
<td>71,343</td>
</tr>
<tr>
<td>Grants receivable</td>
<td>148,500</td>
</tr>
<tr>
<td>Pledges receivable (temporarily restricted)</td>
<td>15,267</td>
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<tr>
<td>Deposits – donor-advised funds</td>
<td>21,998</td>
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<tr>
<td>Fixed assets, net</td>
<td>13,813</td>
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<tr>
<td>Other assets</td>
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<tr>
<td><strong>TOTAL ASSETS</strong></td>
<td><strong>$3,273,140</strong></td>
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LIABILITIES AND NET ASSETS

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<th>Description</th>
<th>Amount</th>
</tr>
</thead>
<tbody>
<tr>
<td>Liabilities</td>
<td></td>
</tr>
<tr>
<td>Accounts payable, grant equipment</td>
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<tr>
<td>Accounts payable and accrued expenses</td>
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<tr>
<td>Net assets</td>
<td></td>
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<tr>
<td>Unrestricted</td>
<td>2,938,936</td>
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<td>Temporarily restricted</td>
<td>15,267</td>
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<td><strong>TOTAL NET ASSETS</strong></td>
<td><strong>2,954,203</strong></td>
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<tr>
<td><strong>TOTAL LIABILITIES AND NET ASSETS</strong></td>
<td><strong>$3,273,140</strong></td>
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Statement of Activities

REVENUE AND OTHER SUPPORT

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<tr>
<td>Contributions</td>
<td>$3,833,716</td>
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<td>Grants</td>
<td>148,500</td>
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<td>Donated services</td>
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<td>Investment income</td>
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<tr>
<td>Realized gain (loss) on sale of stocks</td>
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<tr>
<td>Unrealized gain (loss) on donor-advised funds</td>
<td>544</td>
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<tr>
<td>Net assets released from restrictions, pledges collected</td>
<td>57,500</td>
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<tr>
<td><strong>TOTAL REVENUE AND OTHER SUPPORT</strong></td>
<td><strong>4,064,743</strong></td>
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EXPENDITURES

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<th>Description</th>
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</thead>
<tbody>
<tr>
<td>Program expenses</td>
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<tr>
<td>Grants</td>
<td>3,093,000</td>
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<tr>
<td>Grants distributed</td>
<td>300,946</td>
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<tr>
<td>Other program expenses</td>
<td>196,526</td>
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<td>Management and general</td>
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<td>Fundraising</td>
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<td><strong>TOTAL EXPENDITURES</strong></td>
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(DECREASE) INCREASE IN UNRESTRICTED NET ASSETS

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<tr>
<th>Description</th>
<th>Amount</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>(DECREASE) INCREASE IN UNRESTRICTED NET ASSETS</strong></td>
<td><strong>(47,886)</strong></td>
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TEMPORARILY RESTRICTED NET ASSETS

<table>
<thead>
<tr>
<th>Description</th>
<th>Amount</th>
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<tbody>
<tr>
<td>Net assets released from restrictions, pledges collected</td>
<td>(57,500)</td>
</tr>
<tr>
<td>Net discount/amortizations of pledges</td>
<td>945</td>
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<tr>
<td><strong>DECREASE IN TEMPORARILY RESTRICTED NET ASSETS</strong></td>
<td><strong>(56,555)</strong></td>
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CHANGES IN NET ASSETS

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<tr>
<th>Description</th>
<th>Amount</th>
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</thead>
<tbody>
<tr>
<td>NET ASSETS, beginning of year</td>
<td>3,058,644</td>
</tr>
<tr>
<td>NET ASSETS, end of year</td>
<td><strong>2,954,204</strong></td>
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</table>
In 2010 Cure Alzheimer’s Fund distributed $3,094,287 for research supporting 15 projects.

The Alzheimer’s Genome Project™

The Alzheimer’s Genome Project™ is Cure Alzheimer’s Fund’s core research effort. It has the objective of identifying all relevant Alzheimer’s genes that have not yet been discovered, thereby identifying more targets for the development of therapeutic interventions. It is the largest family-based genome scan ever of a single disease. A milestone for this project was achieved in 2008 with the identification of new genes that confer risk for or protection against Alzheimer’s. This accomplishment was recognized by *TIME* magazine and CNN as one of the top 10 medical breakthroughs of the year.

Prior to this project, there were four known Alzheimer’s genes. Now CAF researchers have identified more than 120 candidate genes and already have confirmed the mechanisms of action of several of these, thereby readying them for drug discovery. Twenty-five of the most important of the genes are undergoing detailed translational analysis.

This effort represents some of the most important Alzheimer’s breakthroughs in recent history, as the genes will greatly facilitate the development of effective therapies for the disease. Each newly identified gene offers fresh understanding of the pathology of the disease and holds promise for the development of therapeutic intervention.

Exploring Mechanisms of Neuroserpin Regulation in Alzheimer’s Disease

Our recent studies have shown that the protease inhibitor neuroserpin, which ultimately leads to decreased breakdown of amyloid-beta and its accumulation, is 70 percent higher in the Alzheimer’s-diseased brain as compared with a normal brain. We’ve also shown that deletion of the neuroserpin gene in an Alzheimer’s disease model mouse restores normal cognitive behavior. Therefore, we will study why neuroserpin is up-regulated in Alzheimer’s disease and will screen for small molecules that block neuroserpin action. These may be useful therapeutic agents to prevent amyloid accumulation and cognitive loss in Alzheimer’s disease patients.
Modulation of Abeta Deposition by Cell-Specific Mechanisms

Sangram Sisodia
University of Chicago
June 2010 – $100,000

This project will employ a genetic strategy that allows for spatial, temporal and cell-specific excision of a mutant PS transgene in order to examine cell-autonomous vs. non-cell-autonomous mechanisms of Abeta deposition. Researchers have strong preliminary studies to validate that the excision strategy works efficiently in mice and have the requisite cellular, biochemical and molecular tools to carry out the proposed studies. If specific cell types in the brain are shown to have an impact on Abeta deposition, investigations will expand to identify the cellular factor(s) responsible for the observed effects, with the notion that strategies aimed at modulating the function of these molecules will be therapeutically efficacious in patients with Alzheimer’s disease (AD).

Design and Development of Novel ACAT Inhibitors for Alzheimer’s Disease

Dora Kovacs
Massachusetts General Hospital
June 2010 – $100,000

The third part of a study focusing on enzyme inhibitors that prevent or decrease the production of Abeta in the brain. This research explores the effectiveness of a drug approved for other uses.

Sustaining Support for the AlzGene

Lars Bertram
Max Planck Institute
December 2010 – $122,336

Cure Alzheimer’s funds the management and continued development of the Web-based database AlzGene.org. The AlzGene database is a revolutionary resource for Alzheimer’s researchers providing data and meta-analyses from hundreds of genetic association studies in an easy-to-use, searchable, Web-based database. This site allows researchers to post their findings and comment on other findings. It aggregates huge amounts of information that will help in the search for the genetic makeup of Alzheimer’s. Currently, the database has more than 1,000 studies. The site also contains a continuously updated list displaying the genes most strongly associated with AD (see “AlzGene Top Results”). Scientists interested in a particular gene can search for it in AlzGene.org to see what previous studies have been reported, receiving a wealth of information in a very short amount of time.
Novel Soluble Gamma-Secretase Modulators

Building on *in vitro* characterization of a novel series of soluble gamma-secretase modulators (SGSMs) funded by Cure Alzheimer’s Fund, the current project is a thorough pharmacological or *in vivo* examination of these molecules to identify the best or “lead” drug candidate.

August 2010 – $250,000

Abeta as an Anti-Microbial Agent

This project explores a completely new and potentially very significant theory concerning the cause of Alzheimer’s disease. The “Anti-Microbial Hypothesis” theorizes that Abeta has a positive function—protecting the brain from invading microbes and defending against bacteria. Initially this project was exclusively funded by CAF, but the subject now is funded also by a multimillion-dollar grant from the National Institutes of Health.

September 2010 – $300,000

Peek and Treat Approach to Diagnosis, Treatment and Monitoring of Alzheimer’s Disease

The objective of this project is to develop a nanotechnology platform that can simultaneously be used to diagnose and treat Alzheimer’s. The work will explore how to develop multifunctional nanocarriers that contain both an imaging agent and a therapeutic agent and also will improve the binding capabilities of the nanocarrier, allowing regulation of the release of therapeutic molecules to amyloid lesions.

May 2010 – $40,000
Mouse Model for Non-Generic Alzheimer’s Disease

Frank La Ferla
University of California, San Diego
October 2010 – $63,851

The SAMP8 mouse model line is a model of accelerated senescence and, interestingly, develops sparse amyloid plaques, which is remarkable given the less amyloidogeneic properties of murine Aβ. Hence, the research hypothesis for this project is that by introducing the more amyloidogenic human APP at the endogenous locus, researchers will be able to more robustly mimic the onset of sporadic Alzheimer’s disease, and thereby utilize this model for pre-clinical development and for widespread distribution to other investigators in the field.

Curcumin Collaborative Project

William Klunk
University of Pittsburgh
Rudy Tanzi
MassGeneral/Harvard

September and October 2010 – $400,000

This collaborative project will identify and characterize novel curcumin-like derivatives for the treatment and prevention of Alzheimer’s disease. The purpose of the study is to develop means of overcoming obstacles to rapid breakdown and creating methodologies for precisely delivering curcumin derivatives to appropriate locations within the brain.

Passive Tau Immunotherapy for Treatment of Alzheimer’s Disease

Virginia Lee
University of Pennsylvania
October 2010 – $100,000

We propose a series of studies to rationally select candidate monoclonal antibodies (mAbs) that bind and neutralize transmissible misfolded tau species, with confirmatory testing of selected mAbs in a mouse model of pathological tau transmission. One or more selected mAbs subsequently will be tested in the established PS19 tau transgenic (Tg) mouse model, which develops forebrain tau inclusions, neuron loss and cognitive deficits, to determine whether passive immunization results in an attenuation of tau-mediated neuropathology.
Within the past five years, late-onset Alzheimer’s disease and Type 2 diabetes have been independently linked with the gene for a protein called SorCS1. This project’s objective is to build on this early data to first better understand the link between AD and T2D, and second to point to new potential drug targets for treating both AD and T2D.

Role of NMDA Receptor in Aβ-driven Synaptic Depression

The traditional signaling induced by NMDA-Rs is triggered by the Ca2+ ions that pass through the NMDA-R channel. However, preliminary data indicate that blockers at the NR2B subunit, but not the NR1 subunit or the channel of the NMDA receptor, prevent the synaptic effects of the NMDA receptor. These findings suggest that Abeta employs novel, non-ion flux NMDA-R signaling mechanisms to produce synaptic depression. These results may open up a new group of potential therapeutic targets for treatment of Alzheimer’s disease.

Effects of Anti-APOE Antibodies on Alzheimer’s Pathology

In this project, the researchers hypothesize that targeting apoE, a component of amyloid plaques, can result in less Aβ aggregation in the brain and decreased Aβ-related pathology, and that this treatment will have fewer side effects than the use of anti-Aβ antibodies. The project will test this hypothesis in this proposal in the context of human apoE isoforms.
Morby Presents at Milken Institute
Global Conference—April 2010

Cure Alzheimer’s Fund Chairman and Co-Founder Jeff Morby presented “Alzheimer’s Disease: Meeting the Challenges in an Aging Society.” Discussion focused on building a climate for a cure, from public awareness to government funding to private investment, and all agreed the creation of a national strategy around research for a cure is critical.

Running4Answers

Founders Carolyn Mastrangelo and Barbara Geiger turned an inspired idea into a wonderful event in New Jersey on April 10, showing the impact individuals can have in the search for a cure. More than 100 runners participated in the four-mile race to benefit Cure Alzheimer’s Fund. The event was an enormous success and we thank Barbara and Carolyn for their vision and tireless effort toward finding a cure. The successful race inspired the second annual event on April 2, 2011.

McCance Presents at Venture Summit East—June 2010

Henry McCance, a Cure Alzheimer’s Fund co-founder and Greylock Partners chairman emeritus, was a keynote speaker at the Venture Summit East 2010. Henry’s presentation featured ideas about how to use a venture capital approach to solve world problems. He focused on Alzheimer’s, citing the scope of the problem and Cure Alzheimer’s Fund’s innovative and targeted approach to end the disease. Venture Summit East features the most influential institutional investors, venture capitalists, corporate buyers, investment bankers and research analysts in the Eastern United States in keynote presentations and panel debates.

Hay Harbor Tennis Tournament—July 2010

Many thanks to all the participants, the Hay Harbor Tennis Club and Diana Fiske for organizing this annual tennis tournament! Using a creative format, this unique tournament raises money for Cure Alzheimer’s Fund.
Webinar—Working Toward a Cure for Alzheimer’s: Clues From Our Genes—July 2010
Our second annual live webinar was conducted over the Internet and featured Dr. Rudy Tanzi’s recent work and progress on the path to a cure. In particular, he focused on how his genetic work is leading to better understanding of the mechanisms behind the disease and leading the way to possible therapeutic intervention.

Tanzi and Armour Present at the White House—September 2010
Cure Alzheimer’s Fund Research Consortium Chair Dr. Rudolph Tanzi and CAF President Tim Armour participated on a scientific panel at the White House. Panelists agreed more funding needs to be invested in finding a cure and better treatments, and that more aggressive efforts at creating public-private partnerships to provide focus for research efforts is crucial. All agreed the nation cannot afford to wait, and the development of effective therapies to prevent or stop Alzheimer’s has to be a national priority, backed by a clear strategy and resources to implement it.

Plan to End Alzheimer’s by 2020 Presented at TEDMED—October 2010
Henry McCance and Rudy Tanzi were invited to the renowned TEDMED conference held in San Diego. TEDMED is a medical technology and health care conference celebrating quality conversation as it relates to personal and public health and is celebrated as the place where health care and medicine collide with brilliant minds and uninhibited imagination. Tanzi and McCance presented together and spoke about the mounting problem of Alzheimer’s, why the current research funding system isn’t moving as fast as it could, and outlined an aggressive plan for ending the disease by 2020. As a result of the presentation, their optimistic and strategic plan was cited by CNN.com and The Wall Street Journal.

The 7 Summits Climb for Alzheimer’s: Memories are Everything—November 2010
The 7 Summits Climb for Alzheimer’s is a yearlong challenge by mountain climber and Alzheimer’s disease advocate Alan Arnette to scale the 7 Summits, the highest peaks on each continent. Arnette climbs in memory of his mother, who passed away in 2009 from Alzheimer’s disease. He chose to climb the 7 Summits to raise awareness of the impact of this debilitating disease and $1 million to advance Alzheimer’s research. He kicked off his campaign with a summit of Mount Vinson in Antarctica. Follow his progress on our website, www.curealz.org, as he travels the world and attempts this ambitious goal.
Cure Alzheimer’s Fund received gifts in honor or in memory of the following in 2010:

George Adams and Sarah Gottlieb
John Adzick
Janice Allen
Norma Alsterlind
Frank Amann
Alan Arnette
Ida Arnette
Henry Barjon
John Bell
Ande Birbaum
Lou Biscaldi
Molly Blau
Barbara Blauvelt
Gail Bradley
Juergen Braun
Richard Bremer
Agnes Tait Buchanan
Nancy Cahners
Merrel Caire
Helen Campbell
David Cantrell
Lil Cantone
Thomas P. Cawley Jr.
Albert Cecil
Richard Charles
Gertrude Cohen
William Crozier
Pauline David
Greg Davis
Mary DeRoy
Juliet DiGangi
George Ditchfield
Albert Drinkwater
Edna Durigon
Margaret Dwenger
Priscilla Eastman
Kerry Elder
Elaine Engelberg
Florence ‘Gene’ Fahs
Helene and Edward Faneuil
Vincent Farinella
Bill Farnsworth
Lawrence Gelfand
Oliver (Sonny) Gentry
Anthony Giorgione
Rolf Goderstad
Brenda Gottlieb
Phyllis Handler
Ed Harris
Robert Harris
John James Hawboldt
Beverly Heath
Mickie Hilbert
Patricia Lee Hild
Allan Holtzman
Florence Homiak
Constance Joy Horlick
Katherine Huguly
Lewis Jackson
Lil Jensen
Virginia Johnson
Olivia Karcher
Henry Karolikiewicz
Joan Kater
Gerald Kean
Dixie Kincaid
Therese Krause
Frank Kromka
Dave and Jo LaCourse
Elsie Lehew
Rafael Linares
Alan Lutchnick
Jack Madigan
Robert Mantica’s father
Mavis Martin
Filippa Marullo
William McBlaire
Ellen McCance and Patrick Pinschmidt
Billy McCook
Gerard McDonald
RoseMarie McDonough
Elizabeth J. McNally
Patricia Minnick
Virginia Wood Motley
Ora Murdock
Joseph Needham
Alice Nelson
Charles Newbrand
Nancy Nieland-Fisher
Ellen Nieman
Paul Nippes
Gerry and Miles Nogelo
Butch Noonan
Glenda Oates
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