

ACHIEVING BREAK THROUGHS

ADVANCING CURE-FOCUSED RESEARCH

The milestone achievements of the Cure Alzheimer's Fund-supported Alzheimer's Genome Project™ mark substantial progress toward ending Alzheimer's disease.

The challenge is to maintain momentum and continue to provide the fast, flexible funding researchers need to find a cure as quickly as possible. It is critical to capitalize on recent research success.

Alzheimer's presents an urgent crisis to our country. If left unchecked, Alzheimer's alone could bankrupt Medicare and Medicaid within the next decade. Alzheimer's is the only disease of the "big seven" (heart disease, cancer, stroke, diabetes, osteoporosis, arthritis and Alzheimer's) to be increasing in number and mortality rates. There is no accounting for the untold mental and emotional anguish the disease causes to patients, caregivers and families.

Cure Alzheimer's Fund is going to the heart of the problem by supporting fundamental research needed to better understand the disease and as quickly as possible develop therapeutic intervention.

Our cover...

features the DNA sequence of the novel Alzheimer's gene GWA-14q discovered in our Alzheimer's Genome Project™, and also illustrates the shape of the now-classic DNA double helix to emphasize the importance of understanding the fundamental genetics of Alzheimer's disease in order to cure it.

Genes are the basic units of heredity and determine the instructions used in the development and functioning of all organisms.

A genetic sequence is a succession of letters representing the order of molecules or nucleotides that form a particular gene. The sequences are made up of four possible nucleotide bases—adenine, cytosine, guanine and thymine—always abbreviated by letters A, C, G and T. These four compounds, ACGT, compose all our genes, which the Human Genome Project™ has estimated to number between 20,000 and 25,000 in humans.

GAGGACAAGTCTGGGAATTA

is a portion of the DNA sequence that composes the GWA-14q gene. The GWA-14q gene was the top finding of the Alzheimer's Genome Project. This gene now joins the four previously established Alzheimer's genes, which have been known for more than a decade and are linked to the production of Abeta 42, the peptide believed by most researchers to be at the core of Alzheimer's pathology.

Cure Alzheimer's Fund has supported the Alzheimer's Genome Project to identify all genes related to risk for Alzheimer's. A major milestone in this project was achieved during the summer of 2008 when Dr. Rudy Tanzi and his colleagues at Massachusetts General Hospital and Harvard Medical School released a paper in the *American Journal of Human Genetics* documenting four new Alzheimer's genes. His team has identified and is confirming the Alzheimer's relationship to more than 70 additional genes that contribute to or provide protection against Alzheimer's disease.





LETTER FROM THE CHAIRMAN
Jeff Morby

Our research is achieving breakthroughs.

2008

was a year of significant progress for Cure Alzheimer's Fund, thanks to the dedicated work of our researchers and generous support of our donors.

When we formed the foundation four years ago, we and our scientific collaborators developed a Roadmap, a research strategy designed to find a cure as soon as possible. The Roadmap had two main components:

- 1. The Alzheimer's Genome Project (AGP)[™]:** The world's largest disease-specific genomic analysis ever undertaken, involving a database of more than 1,700 Alzheimer families and more than 5,600 subjects, designed to identify all human genes responsible for causing Alzheimer's disease. The reason for this project was that Alzheimer's is fundamentally a disease that is strongly influenced by one's genetics, and prior to our AGP, the scientific world had identified only a small fraction of the genes involved in causing the disease. Without the raw material for drug discovery (all Alzheimer's genes), scientists attempting to understand and cure the disease had to focus on trial-and-error approaches and approaches focused only on the four known genes, accounting for a relatively small fraction of the genetics. But we now have made tremendous progress in solving this problem:
 - We have completed the first phase of the AGP and have identified most of the remaining Alzheimer's genes, which represent more than 70 genes implicated in Alzheimer's disease. Of these, approximately 10 are the most important for drug discovery. During the second phase of the AGP our focus will be on identifying additional genes not pinpointed in phase one, and, in parallel, thoroughly investigate the newly discovered AD genes and use this information to develop novel therapies, working with pharmaceutical companies and other collaborators to find potential cures as soon as possible.

Our research is achieving bre

- We announced in December four of the most important genes we had discovered, and as a consequence, our discovery was selected by *TIME* magazine and CNN as representing one of the “Top 10 Medical Breakthroughs” in the world in 2008.
2. **Translation and other studies of the four previously known genes:** Our Research Consortium Chairman, Dr. Rudy Tanzi, always has been a pathfinder in the discovery of Alzheimer’s genes. He co-discovered three of the four genes known before the AGP results, and the characteristics of these genes have permitted scientists to gain a significant, but incomplete, understanding of some of the deleterious causes of Alzheimer’s. Dr. Tanzi and his collaborators are, therefore, in an excellent position to continue to perform research on those genes discovered first, which they are doing. In this regard:
- We have generated an explosion of published work from our funded research. Cure Alzheimer’s Fund work has resulted in 32 papers (12 published in 2008 and listed on page 7). Each of the papers described is a product of research funding provided by us, and each provides further insights into the causes of Alzheimer’s disease.
 - We have co-funded with another non-profit a state-of-the-art process for drug discovery. One of our Research Consortium members, Dr. David Holtzman of Washington University in St. Louis, has developed a unique technique, using transgenic mice with Alzheimer’s disease, for rapidly assessing the cure potentials of different drugs and therapies. He has been carrying out such tests since 2007 and will continue to do so, as we provide him with additional insights coming out of the AGP and other analyses we are carrying out.
 - Our funded oligomer project carried out by six leading universities has disproved one leading theory about how Abeta oligomers bind together and disrupt neuronal signals in the brain and has suggested several novel perspectives as to how oligomers contribute to such disruptions.

**Alzheimer’s
Genome Project™
named one
of Top 10 Medical
Breakthroughs
of 2008 by *TIME*
magazine.**

“There is no cure, no vaccine and no way to diagnose Alzheimer’s disease without an autopsy. But there may be hope in the discovery of four new genes that contribute to the most common form of the disease. ...each newly discovered gene represents a new target and new hope for future drug treatment.”

—*TIME* Magazine,
December 16, 2008

akthroughs.

In addition to the above, the following are projects we undertook in 2008:

Collaboration with other Alzheimer's organizations: Cure Alzheimer's Fund is working with the Alzheimer's Association on the Tomorrow's Leader Award to inspire and enable innovative research by early career researchers. We also are participating in a broad consortium of government agencies and non-profit and pharmaceutical companies, called the Alzheimer's Disease Neuro Imaging Project, to identify appropriate "biomarkers" for Alzheimer's diagnosis and testing.

AlzGene international cooperation: This year our research funding has expanded internationally. We're funding www.AlzGene.org through the Max Planck Institute in Berlin, Germany. AlzGene is the largest database in the world of Alzheimer's scientific research, which we and another non-profit pioneered in the United States. Max Planck Institute has taken on the responsibility of continuing to maintain that database, and we are co-funding that activity.

Going Forward

Now that we have the "raw material" for finding a cure (novel Alzheimer's genes), our activities going forward will focus on how to take full advantage of this newfound knowledge. As mentioned, there are approximately 10 genes that are the most important in terms of their impact (negative and positive) on Alzheimer's and their suitability for driving drug discovery. We are concentrating our activities on those important genes (and others we may further identify in the second phase of the AGP analyses), with the objectives of: (1) understanding how they code for proteins and what the proteins do; (2) understanding the interrelationships among the Alzheimer's genes; (3) conceiving of potential therapies; and (4) testing such potential therapies in the laboratories and with transgenic mice.

Importantly, we do not plan to undertake these projects alone. We already are establishing collaborations with other outstanding scientific organizations, with the objective of bringing in those organizations to assist us to leverage the valuable knowledge we have acquired through the AGP and other funded research.

Obviously, we are far closer to a cure than we were several years ago, and we finally have made the breakthroughs that should lead us to important therapies. We now need to take full advantage of such breakthroughs, but the funding needs to do so will be significant.

We hope you will continue to be a part of this research and help us to carry out our singular quest: to find a cure for Alzheimer's disease as soon as possible.

Many, many thanks for your support.

Jeff Morby
Chairman

Research Progress in 2008

Our funded projects follow our Research Roadmap, which provides a focused strategy for the most effective and efficient route to slowing, stopping or reversing Alzheimer's disease. Our Roadmap is developed and updated by our Research Consortium and Scientific Advisory Board of world-renowned researchers to ensure our research is making progress.

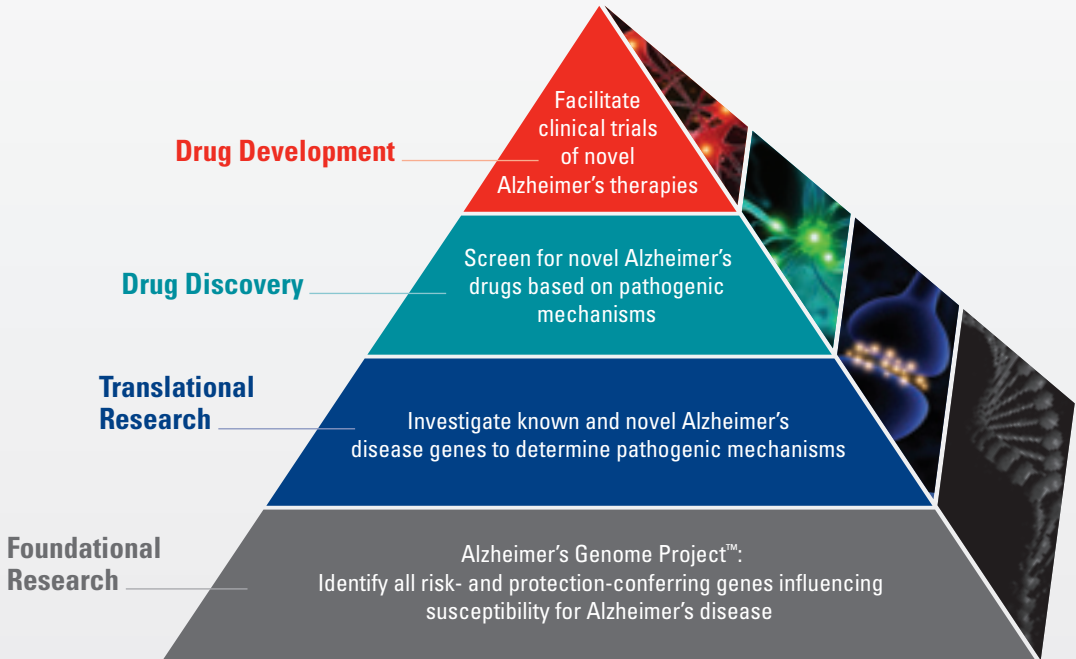
The Research Roadmap Pyramid emphasizes the identification of a broad base of Alzheimer's candidate genes prioritized through the investigative process to those with the highest probability for reliable prediction of Alzheimer's as well as the development of therapies that can prevent disease progression.

Foundational Research begins with the identification of dozens of genes influencing susceptibility for the disease.

Translational Research focuses on which biochemical pathways are impacted by the highest priority Alzheimer's-associated gene defects.

Drug Discovery is guided by the genes and pathways identified through Translational Research as being most amenable for drug development and leads to "compounds" that look most promising for further development.

Drug Development takes those most promising compounds to the next pre-clinical and finally clinical tests for approval for manufacturing and distribution.



To date, our funded projects have been grounded mostly within the Roadmap category of foundational research and have focused on the first step of identifying genes involved with Alzheimer's. We have funded translational research and drug discovery efforts and, increasingly, our work will move up our Roadmap Pyramid as we build on information from the new genes.

Cure Alzheimer's Fund Projects Are Accelerating Progress Toward a Cure

In 2008, Cure Alzheimer's Fund distributed \$2,289,000, supporting 11 research projects, and funding 10 major researchers (and their assistant researchers and lab technicians) at seven national and international leading Alzheimer's research institutions.

Our focus and major accomplishment of 2008 was the completion of phase one of the Alzheimer's Genome Project™ that resulted in the identification of novel genes that affect risk for Alzheimer's disease. This critical work in foundational research offers new information and follow-up potential in better understanding Alzheimer's disease. Each discovery provides hope for a cure and new targets for drug treatment.

Other funded work in 2008 included exploring the role of Abeta oligomers, continued progress of AlzGene.org, support of a microdialysis drug discovery program, exploration of a particular drug targeted at preventing or decreasing Abeta and work on the relationship of traumatic brain injury (TBI) and stroke with Alzheimer's.

Published Papers in Leading Scientific Journals, 2008

The inhalation anesthetic desflurane induces caspase activation and increases amyloid-beta protein levels under hypoxic conditions.

Zhang B, Dong Y, Zhang G, Moir RD, Xia W, Yue Y, Tian M, Culley DJ, Crosby G, Tanzi RE, Xie Z
Journal of Biological Chemistry, May 2008; 283(18):11866-75.

Isoflurane-induced caspase-3 activation is dependent on cytosolic calcium and can be attenuated by memantine.

Zhang G, Dong Y, Zhang B, Ichinose F, Wu X, Culley DJ, Crosby G, Tanzi RE, Xie Z.
Journal of Neuroscience, 2008; 28:4551-60

Amyloid-beta dynamics correlate with neurological status in the injured human brain.

Brody DL, Magnoni S, Schwetye KE, Spinner ML, Esparza TJ, Stocchetti N, Zipfel GJ, Holtzman DM.
Science, 29 August 2008 321: 1221-1224

Oligomeric amyloid-beta peptide disrupts phosphatidylinositol-4, 5-bisphosphate metabolism.

Berman DE, Dall'Armi C, Voronov SV, McIntire LBJ, Zhang H, Moore AZ, Staniszewski A, Arancio O, Kim T, Di Paolo G.
Nature Reviews Neuroscience, 2008; 9:768-78.

Evaluation of the potential excess of statistically significant findings reported genetic association studies: application to Alzheimer's disease

Kavvoura FK, McQueen MP, Khoury MJ, Tanzi RE, Bertram L, Ioannidis JPA.
American Journal of Epidemiology, Oct. 2008; 168:855-865.

Thirty Years of Alzheimer's Disease Genetics: Systematic Meta-analyses Herald a New Era.

Bertram L, Tanzi RE
Nature Reviews Neuroscience, Oct. 2008; 9, 768-778

Association of GSK3B with Alzheimer's Disease and Frontotemporal Dementia.

Schaffer B, Bertram L, Miller L, Mullin K, Weintraub S, Johnson N, Bigio E, Mesulam M, Wiedau-Pazos M, Jackson G, Cummings J, Cantor R, Levey A, Tanzi RE, and Geschwind D.
Archives of Neurology, Oct. 2008; 65:1368-1374.

Genome-wide association analysis reveals novel Alzheimer's disease susceptibility loci in addition to APOE.

Bertram L, Lange CL, Mullin K, Parkinson M, Hsiao M, Hogan MF, Schjeide BMM, Hooli B, DeVito J, Ionita I, Jiang H, Laird N, Moscarillo T, Ohlsen KL, Elliott K, Wang X, Hu-Lince D, Ryder M, Murphy A, Wagner SL, Blacker D, Becker KD, Tanzi RE.
American Journal of Human Genetics, Oct. 2008; 83:623-632.

Structural classification of toxic amyloid oligomers.

Glabe, C. G.
Journal of Biological Chemistry, Oct. 2008; 283, 29639-29643

No association between CALHM1 and Alzheimer's disease risk.

Bertram L, Schjeide BM, Hooli B, Mullin K, Hiltunen M, Soininen H, Ingelsson M, Lannfelt L, Blacker D, Tanzi RE.
Cell, Dec. 2008;135(6):993-4.

The common inhalation anesthetic isoflurane induces caspase activation and increases amyloid beta protein levels in vivo.

Xie Z, Culley D, Dong Y, Zhang G, Zhang B, Moir R, Frosch M, Crosby G, Tanzi RE.
Annals of Neurology, Dec. 2008; Volume 64 Issue 6

The ACAT Inhibitor CI-1011 Reverses Diffuse Brain Amyloid Pathology in Aged hAPP Mice.

Huttner HJ, Utter-Paier B, Barren C, Peach C, Havas D, Duller S, Xia W, Frosch MP, Windisch M, Kovacs DM.
Proceedings of the National Academy of Science, 2008

To see the full list of papers, please visit www.curealzfund.org.

2008 Research Projects



Alzheimer's Genome Project™ Initiative—\$1,228,000

Our core research project has the objective of identifying all relevant remaining Alzheimer's genes, thereby identifying more targets for the development of therapeutic interventions. A milestone for this project was achieved in 2008 with the identification of 70 new genes that confer risk for or protection against Alzheimer's. 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.

Dr. Rudolph Tanzi
Massachusetts General Hospital

Dr. Lars Bertram (pictured on page 9)
Max Planck Institute, Berlin, Germany

Cure Alzheimer's Fund Oligomer Collaborative

A collaboration of leading Alzheimer's researchers from research institutions across the nation. The projects explore the hypothesis that an abnormal increase in levels of synaptic Abeta, and particularly Abeta oligomers, may lead to synaptic dysfunction, cognitive decline and eventually dementia. This highly innovative collaborative project addresses how Abeta oligomers are formed and which types detrimentally impact synaptic dysfunction and neuronal survival in the brain.

As a result of promising results from the first two years of work in 2006 and 2007, further projects were conducted to explore a broader view of the Abeta synaptic feedback loop.



Dr. Virginia M.-Y. Lee—\$100,000
University of Pennsylvania
Abeta Oligomers in Mouse Models of Alzheimer's Disease



Dr. Sangram Sisodia—\$100,000
University of Chicago
Molecular Mechanisms Underlying Hippocampal Neurogenesis by Familial Alzheimer's Disease-linked Presenilin-1 Variants



Dr. David Holtzman—\$100,000
Washington University
Defining Effects of Physiological Synaptic Activity on Abeta Levels: Implications for Alzheimer's Disease



Dr. William Van Nostrand—\$100,000
Stony Brook University
Modulation of Abeta Assembly and Cytotoxicity by a Fragment of Myelin Basic Protein



Dr. Roberto Malinow—\$100,000
University of California at San Diego
Understanding the Cell Biology Underlying the Effects of Abeta on Synapses



Alzheimer's Gene Database

AlzGene.org is an Internet database and forum available on the Web that allows researchers worldwide to share research work and information. The project gathers and analyzes all published studies and data relating to AD genetics, and provides weekly updates regarding ongoing attempts to identify novel AD genes.

Dr. Lars Bertram—\$125,000
Max Planck Institute, Berlin, Germany



ACAT Inhibitor Study

This study focuses on the effect of a particular drug targeted at a cholesterol-related enzyme (ACAT I), with the objective of preventing or decreasing the production of neurotoxic Abeta in the brain.

Dr. Dora Kovacs—\$100,000
Massachusetts General Hospital



Core Facility for Optimal Management of Amyloid-beta Microdialysis Drug Discovery Program

In collaboration with an anonymous funder, Cure Alzheimer's Fund is supporting development of a facility to measure the concentration of Amyloid-beta in real time in the brain of living mouse models that develop features of AD. The model enables screening for drugs that lower Amyloid-beta directly in the brain in relatively high throughput.

Dr. David Holtzman—\$86,000
Washington University



Traumatic Brain Injury and Stroke Relationship to Alzheimer's Disease

This work investigates the increasingly documented link between TBI/stroke and Alzheimer's disease and is aimed not only at developing effective interventions of that linkage but also a contribution to an understanding of the basic Alzheimer's disease mechanism.

Dr. Giuseppina Tesco—\$50,000
Massachusetts General Hospital/Harvard Medical School
Role of BACE in the Pathogenesis of Alzheimer's Disease Following Traumatic Brain Injury (TBI)



Mitochondria Project

This project examines the role of Abeta oligomers in the lipid bilayers found in mitochondria and hopes to make a contribution toward elucidating what is emerging as the multifaceted pathological mechanisms of the Abeta peptide.

Drs. Robert Moir and Rudolph Tanzi—\$200,000 (*Rudolph Tanzi pictured on page 8*)
Massachusetts General Hospital
The Role of Abeta Oligomerization in the Abeta-mediated Disruption of Lipid Bilayers

2008 Financials (Year Ended Dec. 31, 2008)

Statement of Financial Position

ASSETS	
Cash and cash equivalents	\$2,066,159
Contributions receivable and undeposited funds	616,878
Pledges receivable	165,493
Deposits—donor advised funds	20,533
Fixed assets, net	50,238
Other assets	4,683
TOTAL ASSETS	<u>\$2,923,984</u>
LIABILITIES AND NET ASSETS	
Liabilities	
Accounts payable and accrued expenses	<u>\$71,996</u>
Net Assets	
Unrestricted	2,686,495
Temporarily restricted	<u>165,493</u>
TOTAL NET ASSETS	<u>2,851,988</u>
TOTAL LIABILITIES AND NET ASSETS	<u>\$2,923,984</u>

Statement of Activities

REVENUE AND OTHER SUPPORT	
Contributions	\$3,017,928
Donated services	39,366
Investment income	66,188
Realized gain (loss) on sale of stocks	8,437
Unrealized gain (loss) on donor advised funds	(2,571)
Net assets released from restrictions, pledges collected	<u>127,000</u>
	<u>3,256,348</u>
EXPENDITURES	
Program grants, donations and other program expenses	2,520,036
Management and general	227,093
Fundraising	<u>292,357</u>
	<u>3,039,486</u>
INCREASE IN UNRESTRICTED NET ASSETS	<u>216,862</u>
TEMPORARILY RESTRICTED NET ASSETS	
Pledges—current year	7,500
Net assets released from restrictions, pledges collected	(127,000)
Net discount/amortization of pledges	<u>14,211</u>
DECREASE IN TEMPORARILY RESTRICTED NET ASSETS	<u>(105,289)</u>
CHANGES IN NET ASSETS	111,573
NET ASSETS, beginning of year	<u>2,740,415</u>
NET ASSETS, end of year	<u>\$2,851,988</u>

Cure Alzheimer's Fund
Form 990 is available online.
The complete Audited
Financial Statements are
available by request.

Fundraising 2008

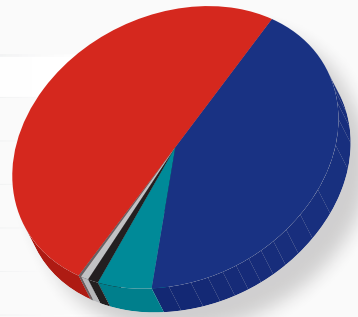
Cure Alzheimer's Fund raises private funds from individuals, corporations and foundations. In this era of declining government support for medical research, private support is essential to maintain progress for an Alzheimer's cure.

In 2008, Cure Alzheimer's Fund raised \$3,184,294 from 1320 donors in cash and in-kind revenues.

Cure Alzheimer's Fund has no endowment and passes all funds raised directly to researchers. The founders of the organization pay all overhead and administrative expenses so 100 percent of donations support research.

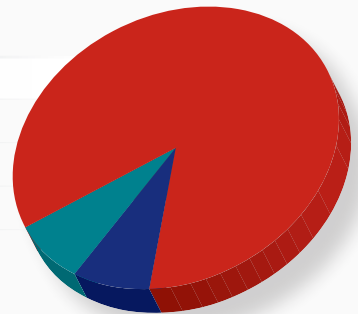
Source of Funds 2008

	Percent	\$ Amount
● Individuals and Family Foundations	50	\$1,597,542
● Founders	42	\$1,341,686
● Bequests	6	\$176,707
● In-Kind Donations	1	\$39,366
● Corporations	1	\$26,629
● Public Foundations	< 1	\$2,364
○ Government	0	\$0
Total		\$3,184,294



Organizational Efficiency 2008

	Percent	\$ Amount
● Research and Programs	83	\$2,520,036
● Management and General Expenses	7	\$227,093
● Fundraising Expenses	10	\$292,357
Total		\$3,039,486



2008 Special Events



Alan Arnette wrapped up a year-long journey of climbing and raising funds for Alzheimer's Disease.

The Road Back to Everest: Memories are Everything

In 2007 and 2008, Alan Arnette took on a challenging goal, a yearlong journey to attempt an Everest summit in 2008 plus raise awareness and funds for Alzheimer's research through Cure Alzheimer's Fund. In preparation, he attempted Denali in Alaska, Shishapangma in Nepal and then summited Aconcagua in Argentina and Orizaba in Mexico and finally returned to Everest in April 2008. While the summit remained elusive on this third attempt, Alan's campaign of fundraising and awareness was impressive. Using a system of a digital camera, PDA and satellite phone, he sent dispatches directly from the climbs, inspiring others to give to research. Alan raised almost \$30,000 and continues to climb and promote giving to Alzheimer's research.

As part of his journey, Alan worked with four schools around the country whose classes followed his progress. We are appreciative for their support and fundraising. Sincere thanks to:

- Andrew Schauer—Webber Junior High School—Ft. Collins, Colo.
- Ginny Allen—Eastern High School—Louisville, Ky.
- Anthony Garcia - Boltz Junior High School—Ft. Collins, Colo.
- Tekla Petrinovich—Straford School—Los Gatos, Calif.



Eastern High School students held an ultimate frisbee tournament to raise money for Cure Alzheimer's Fund.

Brae Burn Country Club Presentation

The Alzheimer's Genome Project™: A Major Milestone Achieved in the Search for an Alzheimer's Cure was presented by Dr. Rudy Tanzi at the Brae Burn Country Club in Newton, Mass. Many thanks to hosts Carl Novotny and Judy Swahnberg.



The Brae Burn Country Club Presentation
From left to right: Henry McCance, Jeff and Jacqui Morby, Carl Novotny, Judith Swahnberg, Rudy Tanzi, Phyllis and Jerry Rappaport

America's Cup

Phyllis Rappaport and Dr. Rudy Tanzi hosted the Cure Alzheimer's Fund yacht in Massachusetts General Hospital (MGH) America's Cup event. Seven guests joined in for an afternoon of racing off Newport, R.I., raising just under \$50,000 for Cure Alzheimer's Fund and part of approximately \$300,000 for MGH.

From left to right: Susan Zises Green, Bridget Baratta, Rudy Tanzi, Katherine Kirk, Phyllis Rappaport, Lois Watson, John Dragat, Mary Campbell and Charles Nolfi



Grampy's Golf

For the third year in a row, Grampy's Charity Open contributed to Cure Alzheimer's Fund. We send our appreciation to Rob Cutler and Jimmy Castle for their continued support. Dates for this year's tournament are July 29–30, 2009.



Hay Harbor Tennis Tournament

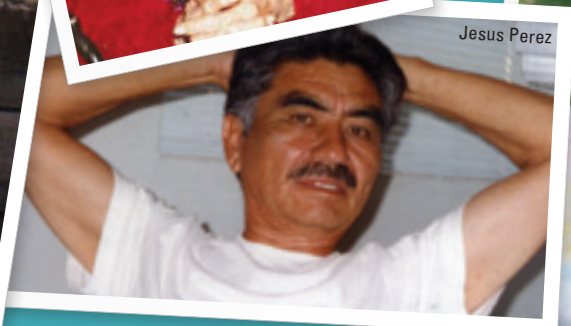
This ladies' tennis tournament—with creative rules like the ability to buy a third serve—raised more than \$6,000 in July for Cure Alzheimer's Fund. Special thanks to the Hay Harbor Tennis Club and Diana Fiske. Date for this year's tournament is July 10, 2009.



If you'd like to host a special event, contact Katie Cutler at kcutter@curealzfund.org.

In Memoriam

Many families choose to honor loved ones by making and requesting donations in memory or honor of a family member. We are grateful to the more than 768 families who have honored Alzheimer's patients and friends with a donation to Cure Alzheimer's Fund.



Why Cure Alzheimer's Fund?

Because Cure Alzheimer's Fund has:

- A focused strategy with one objective: find a cure as quickly as possible.
- World-renowned researchers working together as a team and a Research Roadmap for the most expeditious route to a cure.
- No ownership or intellectual property rights on the research. The return on investment is finding a cure.
- No endowment and all overhead costs are covered by the organization's founders. All donations go directly to research.

We invite you to join our ongoing effort to fund the most promising research to end Alzheimer's disease. And remember, 100 percent of every donation funds research. All overhead costs of the organization are paid for by the founders, so every cent of your gift supports work for a cure.

To make a gift or for more giving information, please visit our website, www.curealzfund.org.



Cure Alzheimer's FUND

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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 #52-2396428.



Cure Alzheimer's FUND

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

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

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

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