Cure Alzheimer’s Fund Proposes a National Research Strategy for Eradicating Alzheimer’s Disease by 2020

With a new administration in Washington and the increasingly urgent threat of Alzheimer’s, Cure Alzheimer’s Fund proposes a national research strategy to accelerate progress toward a cure. The proposal builds on our Research Roadmap, and provides a broader path to intervention by 2020.

We have shared this draft with selected members of Congress and their staffs. We have also worked with the staff of the Alzheimer’s Study Group, a bipartisan effort co-chaired by Newt Gingrich and former Sen. Bob Kerrey, and which includes former Supreme Court Justice Sandra Day O’Connor, Harold Varmus and our own Cure Alzheimer’s Fund.

Cure Alzheimer’s Fund has developed a National Research Strategy for eradicating Alzheimer’s Disease by 2020. This “bottom up” strategy begins with the identification of the largest possible number of genes affecting risk for the disease and increasingly narrows candidate genes, the metabolic systems within which they operate and the proposed therapies for intervention to only those with the highest potential for a cure.

Cure Alzheimer’s Fund supported research has focused to date essentially on the first two blocks of this pyramid.
Henry McCance. It is our goal to work collectively with other funding organizations, the National Institutes of Health and related government agencies to avert the looming crisis of Alzheimer’s. If we can agree on the fundamentals of a national research strategy, we can save millions of lives and billions of dollars within the next decade.

Provided below is a brief excerpt covering the basics of the strategy. Visit our website at www.curealzfund.org to read the proposal in its entirety and a Q&A exploring why it is critical now to address Alzheimer’s at a national level.

National Research Strategy Summary
The overarching goal of the proposed national Alzheimer’s disease (AD) research strategy is to reach a cure by 2020 based on early prediction, pre-symptomatic detection and early prevention. History has shown us this goal will require an acceleration of studies aimed at identifying and investigating all genes that influence susceptibility to AD, placing highest priority on those that provide the most promising biological targets for drug discovery and development.

Studies of the four known AD genes have provided an unprecedented window into the molecular underpinnings of AD. Prior to the discovery of the four known AD genes, the field was limited to merely guessing at the causes of AD with little, if any, success. All four of the known AD genes have pointed to the excessive accumulation of the neurotoxic peptide A-beta in the brain as the primary cause of the disease, although the route to nerve cell death also involves tangle formation. Accordingly, most ongoing clinical trials aimed at modifying disease progression (as opposed to just treating the symptoms) are targeted at lowering A-beta and/or tau accumulation in the brain.

Over the next decade, the influx of funds into AD research should allow for the identification, validation and characterization of all the genes involved in AD susceptibility. These funds also will be implemented to investigate AD-associated defects in these genes. The resulting data can be employed to guide and accelerate novel therapeutics that can prevent AD.

Our strategy includes these steps:
1. Identify all genes contributing to risk for or protection from AD (foundational research);
2. Determine how AD genes contribute to the disease process and which subgroup of genes comprises the most promising therapeutics targets (translational research);
3. Discover therapies that can slow down, stop or reverse AD progress (drug discovery) using the targets provided by AD gene identification and characterization; and
4. Develop a subgroup of the safest and most effective drugs for the treatment and prevention of AD in nationally coordinated clinical trials (drug development).

We propose a national research strategy for AD that will enable the reliable prediction of AD (based on genetics), the early (pre-symptomatic) detection of AD and the development of novel therapies that can prevent the disease process based on the knowledge gained from intensive studies of AD genes. These components will need to dovetail over the coming decade to ultimately enable a pharmacogenomic (personalized medicine) approach to AD, characterized by early prediction, presymptomatic detection and early prevention. With sufficient funding, this could be achieved by 2020.

Visit www.curealzfund.org to learn:
How much is this going to cost?
How much of the strategy already has been accomplished?
What role does the Alzheimer’s Genome Project™ play in the strategy?
A Q&A with President Tim Armour on Cure Alzheimer’s Fund’s Financial Position

Q The news media has been reporting dramatic drops in endowments and in funding for non-profits. How is Cure Alzheimer’s Fund doing?

A Cure Alzheimer’s Fund has no endowment. We keep what funds are left after distribution to research and expenses in money market funds so we can have ready access to money to fund research. Therefore, we are not as exposed to quick changes in the market as many other non-profits are.

We have not experienced the losses that so many non-profits are facing right now. Our mission is to find a cure as quickly as possible, so we invest the donations we receive as rapidly as possible in the research most likely to lead to a cure (as outlined by our Research Roadmap). We do hold some money in reserve for “wild-card,” potentially paradigm-changing discoveries and for unforeseen opportunities to expand our outreach and funding base.

That being said, we are experiencing, as many other non-profits are, a drop in our annual fund-raising—a longer-term downturn in the economy will affect us because it affects our donors. It’s a little too early to tell exactly what the overall picture will be, but we have seen for the period of November 2008 through March 2009 a decrease of about 24% in contributions from the same period last year.

I hasten to add, however, this drop is not significant enough to change our research agenda.

Q Will you be more conservative with your research funding this year?

A In 2009 we will be funding approximately $3 million in research (in 2007 we funded $2.4 million in research and we funded $2.3 million in 2008). We have one objective: to find a cure as soon as we can, and I don’t believe that allows us to be conservative in our funding—frugal, thorough and meticulous, but not conservative.

We were founded by individuals who felt frustrated with the pace of Alzheimer’s research. Our challenge is to move progress forward faster and to do it with relatively short money. We are thrilled that so far we’ve been successful. We often give the baseball analogy that we are trying to find the best players in the game and put the bat in their hands so they can hit a home run. As an example, we gave Rudy Tanzi and his colleagues roughly $3 million and about three years for the first phase of the Alzheimer’s Genome Project™, and they definitely have hit a home run by identifying more than 70 genes that play a part in the disease. That number will probably sift down to a dozen or so “all stars,” but this is the kind of progress we need to keep funding.

Our next step in this project is to find the “best players” who know and understand the genes Rudy and his team have identified and make sure they have a bat.

Q Are there other changes you’ll be making this year as a result of the economy?

A Yes, we will be increasing our spending on our marketing and outreach. Cure Alzheimer’s Fund is unique because all our overhead expenses are paid for by our founders, so we will not be spending donors’ dollars on these initiatives. But we will be investing more in outreach to sustain and grow our donor base beyond the roughly 2,000 families who have so generously supported Cure Alzheimer’s Fund research initiatives during these early years.
Financial Update

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<th>This Quarter</th>
<th>This Year</th>
<th>Inception to date</th>
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<tbody>
<tr>
<td>Fundraising</td>
<td>$191,000</td>
<td>$191,000</td>
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<td>Expenses paid for by the Founders</td>
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<td>Funded Research</td>
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<td>Published Papers</td>
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Research Update

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<tr>
<th>Project</th>
<th>Researcher</th>
<th>Distribution Amount</th>
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<tr>
<td>Relationship of ADAM10 and Dimebon</td>
<td>Dr. Sam Gandy, Mount Sinai Medical School</td>
<td>$100,000</td>
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<td>Understanding the hypothesized relationship between ADAM10, a newly identified Alzheimer’s-related gene and dimebolin, the key ingredient in the anti-Alzheimer’s drug Dimebon.</td>
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<td>MicroRNA’s and APP</td>
<td>Dr. Aleister Saunders, Drexel University</td>
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<td>Test the hypothesis that microRNA’s regulate protein levels of APP and Genome Wide Association Screen (GWAS)-identified levels of APP and GWAS-identified risk genes.</td>
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<td>Specificity and Mechanism of Abeta-Oligomer Action through Prion Protein</td>
<td>Dr. Stephen Strettmatter, Yale University</td>
<td>$100,000</td>
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<td>Part of the Cure Alzheimer’s Fund Oligomer Collaborative.</td>
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<td>Alzheimer’s Genome Project™ Initiative</td>
<td>Dr. Rudy Tanzi, Massachusetts General Hospital</td>
<td>$600,000</td>
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<td>Our core research project; the Alzheimer’s Genome 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 funding continues to confirm, follow-up and prioritize the identified genes for those most likely to guide development of effective therapies.</td>
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Help us fund research with the highest probability of slowing, stopping or reversing Alzheimer’s disease.

Donations can be made through our website www.curealzfund.org or sent directly to our office.

For gifts of securities or direct wire transfers, please contact Tim Armour at 877-CURE-ALZ (287-3259) for further information.
Still Alice
by Lisa Genova, Ph.D.

In her debut novel, now a New York Times bestseller, Lisa Genova describes the onset of Alzheimer’s from the perspective of a 50-year-old woman. Alice Howland, a happily married Harvard professor and mother of three grown children, notices an increasing forgetfulness. Still Alice follows her descent into dementia and Alice’s struggle to maintain her life. Although fiction, Genova has a Ph.D. in neuroscience and beautifully blends characteristics of the disease into this moving and heart-wrenching book. Captivating from the start, the novel is both surprisingly inspiring and terrifying as it details a realistic view of Alzheimer’s.

“An intensely intimate portrait of Alzheimer’s seasoned with highly accurate and useful information about this insidious and devastating disease.”

—Dr. Rudolph E. Tanzi, Cure Alzheimer’s Fund Research Consortium Chairman

I’m Still Here
by John Zeisel, Ph.D.

A Breakthrough Approach to Understanding Someone Living with Alzheimer’s

John Zeisel is an innovator in non-pharmacological approaches to treating Alzheimer’s. His book provides fresh ideas on compassionate care for Alzheimer’s patients. Zeisel shows that during the course of Alzheimer’s, caregivers can have a vibrant and meaningful relationship with people who have the disease. I’m Still Here focuses on connecting with individuals with Alzheimer’s through their abilities that don’t diminish with time, such as understanding music, art, facial expressions and touch. Zeisel demonstrates that people who have the disease are highly creative and emotionally intelligent. By harnessing these capacities, and by using other approaches to treatment—such as building memory cues into their living environment, which encourages independent movement and helps eliminate sources of frustration—it’s possible to offer them a quality life with connection to others and to the world.
Save the Date

Please join us for a Webinar with Dr. Rudy Tanzi
Wednesday, May 20, 2009 from 11 am to Noon

“Progress in the Search for an Alzheimer’s Cure”

This live presentation by Dr. Rudy Tanzi will be conducted over the internet. Each guest will participate through their own computer and phone (or VoIP). The presentation will be interactive and allow questions from the attendees. Dr. Tanzi will talk about recent breakthrough work and progress on the path to a cure.

Dr. Tanzi is the Joseph P. and Rose F. Kennedy Professor of Neurology at Harvard Medical School and the Director of the Genetics and Aging Research Unit at Massachusetts General Hospital. He has been investigating human neurodegenerative disease since 1980 when he participated in the pioneering study that led to location of the Huntington disease gene. He has identified several Alzheimer’s disease genes, including the first Alzheimer’s gene, the beta-amyloid protein precursor (APP). His work in the Alzheimer’s Genome Project, which has identified other new genes, was recognized by Time magazine as one of the top 10 medical breakthroughs of 2008. Dr. Tanzi is a world-renowned leader in studies of Alzheimer’s disease genetics.

Register on our website, www.curealzfund.org or contact Katie Cutler at kcutler@curealzfund.org or 781-237-3800.