Cure Alzheimer's FUND
targeting breakthrough research

Roadmap 2.0

Closing in on Smarter Therapies

We've now made considerable progress …[and have] developed an important integrated view of the entire disease process.

From its very inception, the underlying strategy of Cure Alzheimer's Fund was to help the Alzheimer's research community get on a smarter, faster trajectory toward a cure. To do that, its Research Consortium developed, in 2006, a roadmap with four fundamental planks:

1. **Foundational**—Find all genes that contribute to risk.
2. **Translational**—Use genetic knowledge to discover Alzheimer's true pathology.
3. **Drug Discovery**—Use new understanding to choose/create compounds.
4. **Drug Development**—Partner with pharmacology to develop those compounds.

We've now made considerable progress in all of these areas and have, as a result, developed an important integrated view of the entire disease process. The accompanying article, "Alzheimer's Pathology: An Integrated View," by Cure Alzheimer's Fund Chairman Jeffrey Morby, outlines that integrated view in detail.

In the upcoming Oct. 10 symposium at the Mandarin Hotel in Boston (also streaming live), three members of the Cure Alzheimer's Research Consortium—Steve Wagner, Ph.D.; Sangram Sisodia, Ph.D.; and Chairman Rudy Tanzi, Ph.D.—will flesh out this integrated view and also lay out recent developments and new tactical approaches toward effective Alzheimer's therapies.

Foundational

On the foundational front, Cure Alzheimer's Fund has made enormous progress by spearheading the Alzheimer's Genome Project (AGP)™, the largest family-based single-disease gene scan to date. Out of that project, Rudy Tanzi has identified more than 120 candidate genes and already has confirmed the defects and pathological mechanisms for several of these genes. 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.

Translational

Each newly identified gene offers fresh translational understanding of the pathology of the disease. For example, Sam Sisodia is using a genetic approach to enhance our understanding of how a protein associated with Alzheimer's, called amyloid, is formed in the brain.

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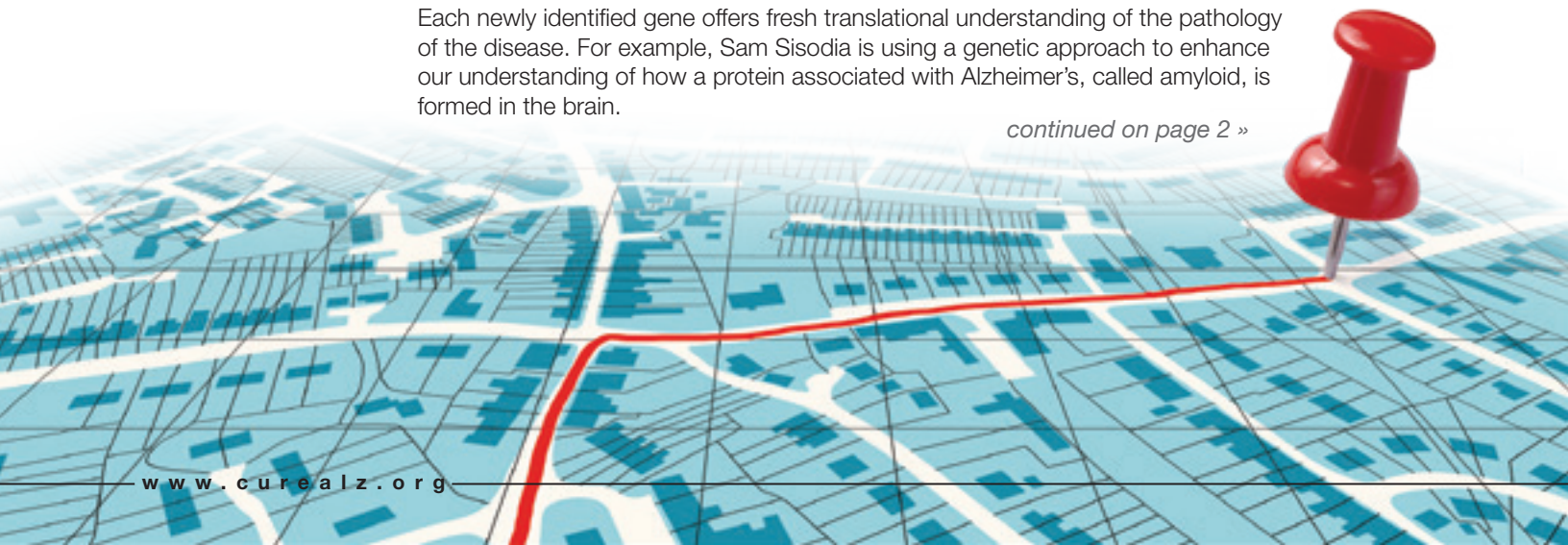

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Drug Discovery and Development

In the realm of drug discovery and development, CAF-funded researchers currently have four promising Alzheimer's drugs/drug groups in pre-clinical development. All three compounds potentially could stop the disease in its tracks:

- **The first drug, Bexarotene,** is studied by Sam Sisodia, Ph.D., of the University of Chicago, Robert Vassar Ph.D., of Northwestern University and David Holtzman, M.D., of Washington University, St. Louis. It already has been approved for use in a rare form of cancer, and recently was found to clear senile plaques in mouse models of Alzheimer's disease.
- **The second group** is composed of anti-beta-amyloid compounds called gamma secretase modulators, being developed by Steve Wagner at the University of California, San Diego and Rudy Tanzi at Massachusetts General Hospital (MGH). This project was selected by the National Institutes of Health for funding in its new "blueprint" program set up by NIH Director Francis Collins; the goal here is to get it into clinical trials in the next two years.
- **The third drug class** is known as ACAT inhibitors. Dora Kovacs at MGH discovered these drugs dramatically reduce plaques in mouse models of Alzheimer's disease. The goal is to get them into clinical trials by 2015.
- **The fourth drug class** is based on recently published findings from Tanzi's laboratory showing that curcumin, the main ingredient in turmeric, lowers the production of the amyloid beta protein. Since curcumin does not enter the brain, Tanzi is working with Dr. Bill Klunk at the University of Pittsburgh to make drugs based on curcumin that will be able to enter the brain and, it is hoped, act as anti-beta-amyloid compounds.

We look forward to a more complete picture presented by Sisodia, Tanzi and Wagner on Oct. 10. ■

Alzheimer's Pathology: An Integrated View

by Jeffrey Morby, Cure Alzheimer's Fund chairman and co-founder

As a result of Cure Alzheimer's Fund research, an integrated view of the causes of Alzheimer's pathology has emerged. That view begins with a concept of what makes up a healthy brain. Abeta, we now know, performs a number of useful functions within the brain. In a healthy brain, moderate amounts of Abeta will be produced and cleared from time to time. Clearance is provided by several proteins, the most important of which are the APOE proteins (of which there are three variants). Hence, the healthy brain maintains a proper equilibrium between the production of Abeta and its clearance—a healthy hemostasis.

In an unhealthy brain, that proper equilibrium is not maintained, and the brain ends up with too much Abeta—allowing Alzheimer's pathology to begin. Too much Abeta in the brain can result from a number of causes, including defective genes. Some genes produce too much Abeta; other genes hinder Abeta clearance from the brain. Some brains are hit by both problems at the same time—over-production and inadequate clearance. This often leads to Alzheimer's disease.

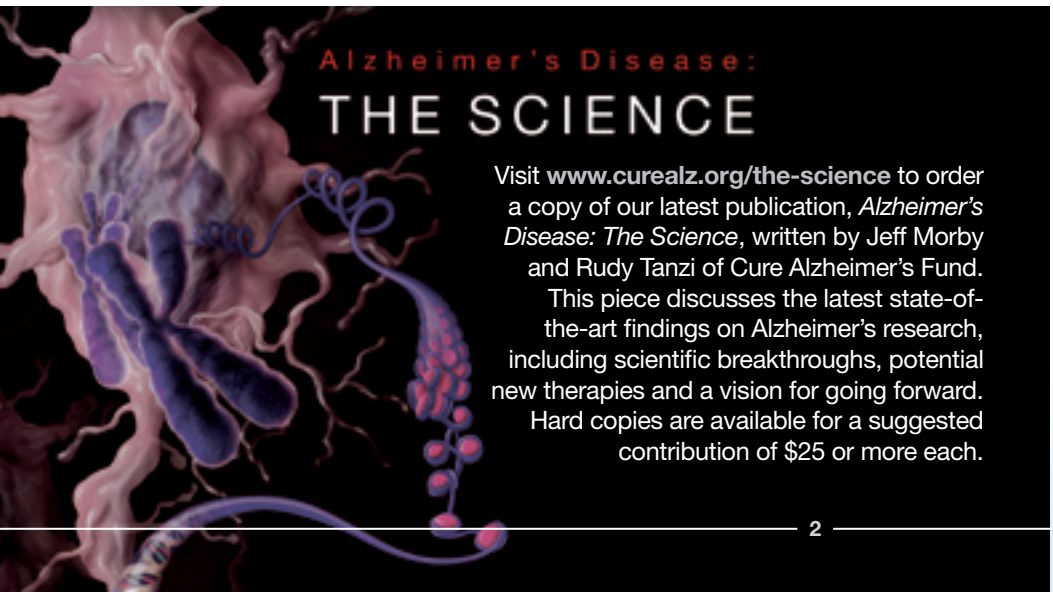
Based on recent evidence, we now believe one of the main functions of Abeta, in its role as part of the innate immune system, is to attack pathogens. In a healthy brain, when invading pathogens are detected, new Abeta is produced to attack and destroy them. Once the pathogens are eliminated, excess Abeta is cleared from the brain, and equilibrium is restored. In the unhealthy brain, too much Abeta may be produced, or it may not be sufficiently cleared, or both. The excess Abeta will then amalgamate into amyloid plaques, which causes inflammation and damages neural cells (and further provokes the creation of more Abeta). Concussions, stroke and other injuries also will provoke the creation of additional Abeta, and may initiate a chain of events leading to Alzheimer's disease.

Tau is the next piece in the integrated portrait of Alzheimer's. Too much Abeta apparently initiates the creation of Tau "tangles," which destroy nerve cells, provoke the creation of more Abeta and inflammation in a vicious feedback loop, and proliferate to other parts of the brain. The processes involved in Tau proliferation are not yet well understood.

To summarize, Alzheimer's pathology requires three elements:

- Too much Abeta, due to excessive generation and/or inadequate clearance;
- Tau tangles, sparked by the overabundance of Abeta leading to neurodegeneration; and
- Inflammation, provoked by too much Abeta and by Tau tangle-induced neurodegeneration, which subsequently produces more Abeta and Tau tangles. One leads to the next and eventually, a vicious cycle begins.

Cure Alzheimer's Fund is tackling all of the above elements. ■



A Fight to the Finish

On March 8, Bobby Zerwick, 22, set out to hike the entire Appalachian Trail (AT). His goal? To raise $5,000 to help in the fight against Alzheimer's—the disease his grandmother and his girlfriend's grandmother suffered from before they passed away.

A recent graduate of Kutztown University with a B.A. in marketing, Zerwick looked at a number of nonprofits before he chose to support Cure Alzheimer's Fund. He selected it because he knew all the money he raised would go directly toward finding a cure for Alzheimer's disease.

Bobby at the 2,000-mile point on the AT

Determination

The AT runs 2,187 miles long through 14 states—from Springer Mountain (3,700 feet) in Georgia to Mount Katahdin (5,200 feet) in Maine—but this is no walk in the woods. While there are long stretches of relatively flat terrain, the AT includes dozens of significant summits, including Mount Moosilauke (4,820 feet), Mount Lafayette (5,200 feet) and Mount Washington (6,200 feet) in the northern part of the trail.

When Zerwick was in North Carolina, his feet hurt so badly he could barely move, and he almost gave up. He had worn the wrong boots and needed to recover for a few days before he could continue. After that he took a few breaks along his journey to recharge, but his determination to finish never wavered—partly because he was cheered on by his family and his girlfriend, Ashley, and partly because that's the kind of person he is.

On July 9, Mike Curren, senior vice president of Cure Alzheimer's Fund, met Zerwick on top of Mount Greylock in Massachusetts. They had lunch together and talked about some of Zerwick's experiences. Then Zerwick asked Curren whether 'there was anything new on the research front with Alzheimer's.' "It was clear to me how personally invested he is in this cause," says Curren.

New challenges

In late July, Zerwick still had New Hampshire and Maine to conquer. This part of the AT presented different challenges from the previous 12 states, such as miles of exposed ridges above the treeline where hikers are tested by the elements, including plummeting temperatures at night, high winds and potentially even snow. But New Hampshire also has a series of huts along the trail where hikers can stop to get a warm meal, restock on essentials (bug spray and batteries), and even sleep in a bunk bed for a nominal cost. The AT in Maine stretches more than 100 miles and is famous for its deer flies and steep drop-offs that require hikers to "scramble" on all fours. To complete his journey, Zerwick had to be prepared for anything.

Throughout his trek Zerwick saw familiar faces along the AT. He carried a 20-pound pack on his back with only the essentials: a tent, sleeping bag, change of clothes, food and water, flashlight, toothbrush and cell phone, which he only used when he had to. He also carried two walking sticks that he says were "essential for hiking downhill."

Zerwick averaged about 15 miles per day over a 10- to 12-hour span, despite the weather. He only stopped when downpours made the trail like a river. He lost at least 15 pounds, got into the best shape of his life, and still was always hungry. He survived on Pop-Tarts®, GORP (a mixture of granola, oats, raisins and peanuts) and other high-energy foods.

Mike Curren and Bobby Zerwick at Bascom Lodge on top of Mount Greylock, Mass.

A double celebration

After more than five months in the wilderness, Zerwick finished the AT on Aug. 17. But Zerwick had something else to celebrate as well. After hiking more than 2,000 miles, his girlfriend Ashley joined him for the very last part of his trek—5,200 feet up Mount Katadhin in Maine. And there on the summit he proposed to her. After so many months in isolation, Bobby is about to embark on a new journey through life with Ashley at his side. "We're so happy for both of them," says Curren.

Zerwick not only finished the AT at a very respectable pace, he exceeded his fundraising goal, raising more than $5,000 for Cure Alzheimer's Fund. "We are grateful for his commitment to our cause," says Tim Armour, chairman and CEO of Cure Alzheimer's Fund. "I'm really proud of what I've accomplished," says Zerwick. "It's rewarding to know that I did something to help other people."

Additional donations are always welcome. Please visit www.firstgiving.com and search for Bobby Zerwick. ■

Bobby proposes to Ashley at the peak of Mount Katahdin

FEATURED RESEARCHER

Steven L. Wagner, Ph.D.

Since the 12 members of Cure Alzheimer's Fund Research Consortium set and lead the direction of research to fulfill our mission, we will feature one member in each issue of our quarterly report. These profiles will tell the story of each of our consortium members—their background, the research projects they're working on and why their research is important to finding a cure. Last quarter we heard from Sangram S. Sisodia. This quarter we are featuring Steven L. Wagner, Ph.D., project scientist, principal investigator, University of California, San Diego School of Medicine, Department of Neurosciences.

Growing up

The oldest of four children, Steve Wagner was born in Louisville, Ky.—home of the Kentucky Derby. Wagner's father, a pharmacist, owned both a veterinary drugstore and Wagner's Pharmacy—an old-fashioned drugstore that has served milkshakes at soda fountains since 1922. Wagner's is the iconic landmark where Bob Costas opened up NBC's broadcast of the Kentucky Derby in 2005 and it's been featured in a number of movies—including "Secretariat."

As a kid, Wagner spent long hours working behind the drug counter while his father filled prescriptions. Wagner was fascinated by all the different pills. "I remember my father getting irritated because I wanted to know what every one of the drugs did exactly. I knew then that I would end up in drug discovery one day," says Wagner. His dream was to work in a medical research laboratory on the beach, and though he didn't know it then, his dream eventually would come true.

Education

After high school, Wagner attended Bellarmine College in Louisville. From there he went to graduate school at the University of Louisville School of Medicine to study molecular biology and biochemistry. There he worked with Dr. Robert D. Gray, a protein and peptide chemist. "I loved working with complex membrane proteins that were critical in metabolizing xenobiotics (drugs)," says Wagner. "My work there provided a good foundation for the types of molecules I'm targeting now in Alzheimer's disease. I knew then that discovering drugs for unmet medical needs was what I wanted to do."

Change of scenery

In 1986, Wagner left Kentucky for the Golden State. He went to the University of California, Irvine College of Medicine for a National Institutes of Health-funded post-doctorate fellowship. "I was interested in working with Dr. Dennis Cunningham and studying the molecules that were recently discovered in his lab," says Wagner. "These proteins, although harboring potent anti-blood clotting activity, weren't bloodborne at all, and they expressed themselves almost exclusively in the brain. I basically followed the proteins into the brain and never left."

Wagner was honored as a Hewitt Medical Research Associate at the UC Irvine College of Medicine, a prestigious award that allowed him to work there for six years in the Department of Microbiology and Molecular Genetics, where he focused his work on Alzheimer's disease. There he and his colleagues had tremendous success. They isolated, for the first time, the amyloid precursor protein (a protein later found to be critical in the pathogenesis of Alzheimer's disease).

Rudy and Sam

In 1987 Wagner met Dr. Rudy Tanzi, Ph.D., from Harvard/Massachusetts General Hospital at a NATO conference in Italy focusing on neurodegenerative diseases and the roles proteases and protease inhibitors play. "I was doing a presentation on protease nexins (proteins that connect proteases to cells)," says Wagner, "and Rudy came up to me afterwards because he was very interested in my research. He said, 'There's something going on here with your protease nexins and my amyloid precursor protein gene,' (Tanzi had cloned the human gene that same year), and sure enough he was right." The protease nexin II turned out to be the amyloid precursor protein, which, like protease nexin I, also was shown to be a robust inhibitor of blood coagulation.

"Rudy and I hit it off right away—we were almost exactly the same age, both athletic and liked jazz." Then Tanzi introduced Wagner to Dr. Sam Sisodia, Ph.D., a cell biologist from the Johns Hopkins University School of Medicine, and the three of them became the best of friends. "When we are together we spend hours talking science and how to get closer to a rational therapeutic approach to Alzheimer's," says Wagner. "I am lucky to work with people I truly enjoy being with."

NeuroGenetic Pharmaceuticals

In 1991, after UC Irvine, Wagner found his lab on the beach at the Salk Institute of Biotechnology/Industrial Associates Inc. (SIBIA) in La Jolla, Calif. He brought in a seasoned pharmaceutical executive, and together, with a number of other molecular biologists and neuroscientists, they transformed SIBIA into a neuroscience drug discovery company (SIBIA Neurosciences). In 1995, they took the company public and in 1999 sold it to the pharmaceutical giant Merck.

Shortly after that, Wagner and Tanzi co-founded a biotech company called NeuroGenetic. There they established a target discovery program based on genetics, which Tanzi led, and a drug discovery program, which Wagner headed up. Sisodia headed their Scientific Advisory Board. "We built a team of geneticists, chemists, biologists, pharmacologists and clinicians. That's where we discovered the original gamma secretase modulators (GSMs)." Wagner worked at NeuroGenetic for almost eight years as its chief scientific officer and says, "It was one of the best times of my life, because it gave me a chance to work closely with both Rudy and Sam."

Gamma secretase modulators

Wagner and Tanzi first discussed the idea of GSMs for Alzheimer's on the tennis court. "We'd play tennis whenever we got together—we even played in New Orleans on a hotel roof," says Wagner. "We said

'Wouldn't it be great if we could reverse the phenotype of familial Alzheimer's disease genes with small molecules?' " and the idea was born.

I think these molecules have a chance of helping a lot of people one day because they modulate amyloid production—they don't shut it down—which causes other problems," explains Wagner. "Instead, they bind one of the two key enzymes that produce amyloid—sticky plaques that riddle the brains of Alzheimer's patients—which results in much fewer plaques. If we administer these at the right time to the right patient populations, it could help to slow down the disease process."

Cure Alzheimer's Fund

In 2004, Cure Alzheimer's Fund was founded and Tanzi and Sisodia joined as members of the Research Consortium. Wagner came on board a few years later. Today, Tanzi is the geneticist in Boston, Sisodia is the cell biologist in Chicago and Wagner is the biological chemist in San Diego. "From coast to coast we've got things covered," says Wagner.

In 2009, Wagner joined the Department of Neurosciences at the UCSD School of Medicine, where he now runs his own laboratory. "Today I'm doing almost the same thing I was doing when I was on the other side of the fence in industry, although I'm working with academicians and the government and as part of the Cure Alzheimer's Fund. I'm doing translational medical research, which means moving molecules from the bench to the bedside—which is quite a task. As a member of the Cure Alzheimer's Fund consortium, I can talk to whomever I need to bounce ideas off of. We have such an elite group of people to exchange ideas with. You're much more confined when you're working in the biopharmaceutical industry—you can talk freely only with the people you work with in your company. Here, it's the best of both worlds."

Getting to a cure

Wagner received two grants from Cure Alzheimer's Fund in 2009 and 2010 to provide "proof of concept" for the innovative GSMs that he, Tanzi and their colleagues developed. Together, they were able to leverage their findings into a blockbuster NIH award as part of the premier "Blueprint" grant program for fast-track development of promising Alzheimer's drugs. Tanzi and Wagner are collaborating on this five-year project, now totally funded by NIH, with the goal of developing a compound that will remove only the toxic amyloid from the brain of Alzheimer's patients. The ultimate goal is to bring drug candidates into Phase I clinical trials.

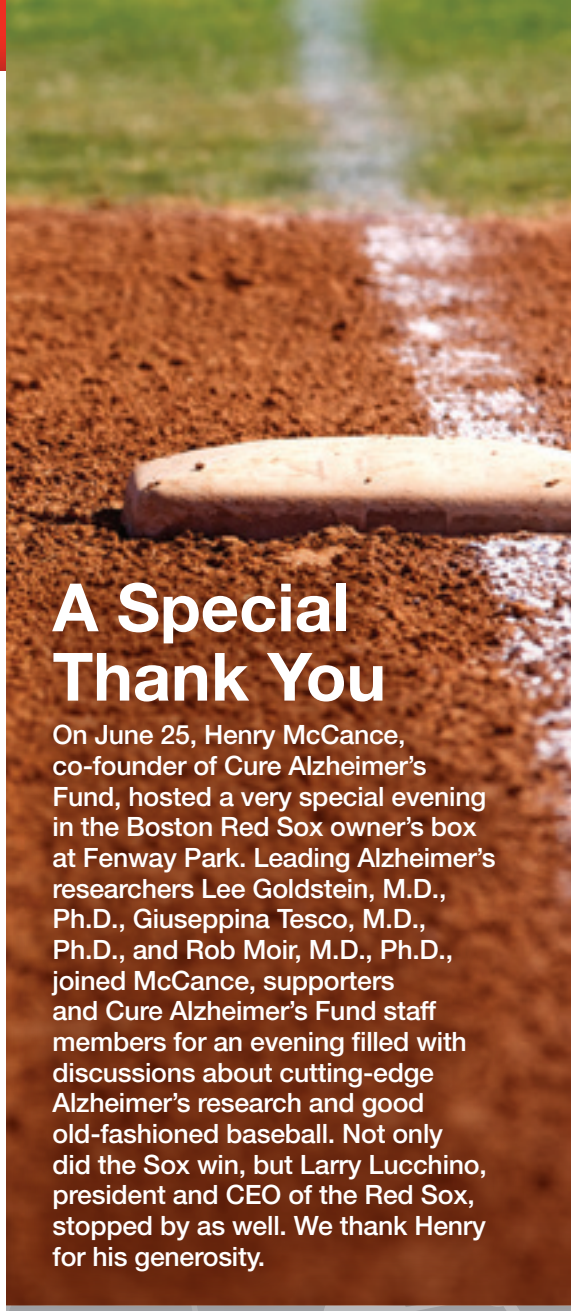
"The Blueprint grant is a huge vote of confidence by the NIH in Steve's work," says Tim Armour, chairman and CEO of Cure Alzheimer's Fund. "They believe that the work Steve's lab is doing has the best chance of becoming an effective Alzheimer's drug of all the research being done today." With the large NIH grant now in place, CAF has provided an additional $150,000 to determine precisely how his compound actually works. "I think this is the final frontier for the beta-amyloid hypothesis," says Wagner. "We may have finally found what we've been looking for."

Personal loss

Wagner's father passed away a few years ago after starting to show signs of dementia. "While my dad never had an autopsy, I'd bet he had Alzheimer's," says Wagner. "He was a diabetic, which has been linked with Alzheimer's, and he would ask repetitive questions and get disoriented towards the end of his life. My father was someone whom I loved very much and respected."

Sadly, Wagner also lost his wife a couple of years ago to a very rare and aggressive form of cancer. "She was only 43, and I think about her every day," he says. "You go through life thinking you're one of the luckiest people in the world and then that happens. My work really helps me get through it."

Dr. Wagner will join his friends and colleagues Tanzi and Sisodia at Cure Alzheimer's Fund's fall symposium on Oct. 10 in Boston. Please join us to hear the latest breakthroughs on the Alzheimer's front. ■



A Special Thank You

On June 25, Henry McCance, co-founder of Cure Alzheimer's Fund, hosted a very special evening in the Boston Red Sox owner's box at Fenway Park. Leading Alzheimer's researchers Lee Goldstein, M.D., Ph.D., Giuseppina Tesco, M.D., Ph.D., and Rob Moir, M.D., Ph.D., joined McCance, supporters and Cure Alzheimer's Fund staff members for an evening filled with discussions about cutting-edge Alzheimer's research and good old-fashioned baseball. Not only did the Sox win, but Larry Lucchino, president and CEO of the Red Sox, stopped by as well. We thank Henry for his generosity.

Help us fund research with the highest probability of slowing, stopping or reversing Alzheimer's disease. Donations can be made through our website, www.curealz.org/donate, 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.

Art for Alzheimer's

After reading Barbara Kingsolver's *Animal Dreams* in Honors English at Scituate High School, Lexie Fidas, 15, was given another class assignment—to create a difference in society, much the way the characters in the book did.

Art for Alzheimer's

Fidas came up with an idea that not only would change the lives of others, but her own as well. Since one of the characters in *Animal Dreams* has Alzheimer's disease, Fidas decided to help local Alzheimer's patients create their own artwork at Riverview Nursing Home, near Fidas' hometown of Hope, R.I. "I thought using art would be a great way to connect with Alzheimer's patients," she says. Although her grandmother had suffered from dementia, Fidas hadn't had any experience with the disease prior to beginning her project.

Creating the artwork

"I included anyone who wanted to participate," explains Fidas. She and her mother set up a drawing section with crayons, colored pencils and pastels, and a painting section with brushes and sponges. "Even though some of the patients didn't know what they were painting, or didn't remember how, they did it," says Fidas. Afterward, she and her mom framed each piece and Fidas auctioned them off at her school. "We raised $80!" adds Fidas proudly.

Cure Alzheimer's Fund

"I decided to give the money to Cure Alzheimer's Fund, because all their donations go directly to research," she explains. "This project was one of the most life-changing things I have ever done. Even though the people at Riverview may not remember me, I will always remember them." ■

Lexie Fidas creates art with Alzheimer's patients

A Test of Endurance

On Sept. 6, Brett Reynolds, age 30, flew to London to swim the English Channel. Reynolds took on this challenge to honor his grandmother, who suffered from Alzheimer's disease before passing away. "I wanted to raise money to help stop the disease and to honor her memory," says Reynolds.

His goal was to raise $5,000 for Cure Alzheimer's Fund. To date he has raised an incredible $7,570! "I wanted to support an organization focused on finding a cure" says Reynolds. "I was impressed with their vision and believe in their entrepreneurial approach."

Originally from Southern California, Reynolds was always an avid swimmer. He grew up playing water polo, was on Division 1 USC's NCAA championship water polo team and played for the U.S. men's team in South Korea in the World University Games.

'The swimmer's equivalent of climbing a mountain'

Only 10 percent of people who set out to swim the channel actually succeed. Stretching 21 miles from Dover, England, to Calais, France, it can take anywhere from seven to 18 hours to swim across. But it's not the distance that makes the swim so difficult.

The channel is the world's busiest seaway, with more than 700 commercial ships and ferries running through it daily. With water temperatures between 59 and 63 degrees and no wetsuits permitted (to make the swim "official"), hypothermia and cramping are severe risks. Still, the biggest threat is the currents, because every six hours or so they change direction. If swimmers get the timing wrong, even if they're in sight of the coast, they might as well give up, because they'll only be swimming backward.

To make the crossing official, swimmers cannot touch their guide boat or another person at any time during their swim to receive food or water, and they must present a passport when arriving in France. Despite all these challenges, seasickness was Reynolds' biggest concern.

The swim

Reynolds began his journey at 3:30 a.m. on Sept. 7 in darkness, with his mom cheering him on from a chartered boat. "She is the only one of my family and friends who doesn't succumb to seasickness," says Reynolds. Swimming the channel is not something that can be done alone, since swimmers need someone to provide them with the essentials: food, water and light, not to mention encouragement.

Brett Reynolds before his channel swim

Three hours into his swim, Reynolds experienced pain in his right shoulder, which unfortunately stayed with him throughout the race. Toward the end, he began to show signs of hypothermia, but seasickness wasn't an issue. "Swimming the channel was a humbling experience," he says. "I never would have made it without the support I received from so many friends and family on (and off) the boat." Reynolds completed his swim in just 12 hours and 5 minutes. "We are thrilled with Brett's accomplishment and incredibly appreciative of his fundraising efforts," says Mike Curren, a senior vice president of Cure Alzheimer's Fund.

Next big adventure

Reynolds lives in San Francisco with his wife and works in commercial real estate consulting and valuation. They are expecting their first child next spring and are very much looking forward to their next big adventure on dry land. ■

Financial Update

	This Quarter	YTD*	Inception to date
Fundraising	$154,204	$1,752,130	$25,144,024
Expenses paid for by the founders	$200,030**	$814,231	$5,363,900
Funded research	$350,343	$1,575,343	$16,296,484

These numbers are as of Aug. 31, 2012

***Numbers shown reflect net expense paid for by the founders after applying $33,445 returned from a prior period grant as the project was canceled before completion.*

Research Update

Research funded during the third quarter of 2012

Project	Researcher	Distribution Amount
The Putative Role of Red Blood Cell CR1 Levels in Amyloid-B Clearance and Alzheimer's Disease	Cynthia Lemere, Ph.D. Brigham and Women's Hospital, Boston	$100,000
The roles of Eps homology domain (EHD) proteins and synaptic activity in axon transport of the Alzheimer's Beta-secretase BACE1 in the brain	Robert Vassar, Ph.D. Northwestern University	$100,000
BACE1 transcytosis in Alzheimer's diesease pathogenesis	Gopal Thinakaran, Ph.D. University of Chicago	$100,000
Replication Studies for Bexarotene	Sangram Sissodia, Ph.D., University of Chicago	$15,615
	Robert Vassar, Ph.D., Northwestern Univesity	$34,728
iPS-derived and trans-differentiated human neurons as models to study Alzheimer's disease	Marc Tessier-Lavigne, The Rockefeller University	$100,000
The role of PICALM in vascular clearance of amyloid-b	Berislav V. Zlokovic, M.D., Ph.D. University of Southern California	$100,000
Alzheimer's Genome Project™	Rudy Tanzi, Ph.D. Harvard Medical School Massachusetts General Hospital	$600,000

An Extra Set of Hands

Madeleine Adelson, a senior at Tufts University this year, joined Cure Alzheimer's Fund as an intern this past summer. "Maddie has helped us with updating our website, finishing key publications, organizing our webinars and preparing for our symposium in October," says Sally Rosenfield, senior vice president of Cure Alzheimer's Fund. As someone who is interested in public relations for nonprofit organizations, Adelson saw the intern position as a great learning opportunity. "At Cure Alzheimer's Fund I've been exposed to many different tasks involved in running a nonprofit," she explains. "While I don't have any personal experience with Alzheimer's disease, I've come to appreciate how vital it is that Alzheimer's research receives more attention and funding. I love working for such an important cause." We thank Maddie for her contributions over these past few months and are grateful she will continue to help out during her senior year at Tufts. ■

Cure Alzheimer's FUND

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Mission

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

Research Consortium

Scientific Advisory Board

Board of Directors

Administration

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.

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Don't Miss Our Fall Symposium

WEDNESDAY, OCT. 10, 4–5:30 p.m.

Taking Control of Alzheimer's Through Research—The Roadmap to Therapies

Leading Cure Alzheimer's Fund researchers Rudy Tanzi, Ph.D., Sangram Sisodia, Ph.D., and Steve Wagner, Ph.D., will gather at the Mandarin Oriental Hotel in Boston to discuss the roadmap to Alzheimer's disease therapies and take questions from the audience. David Shenk, author of the national bestseller *The Forgetting, Alzheimer's: Portrait of an Epidemic*, will moderate the event. Please join us for this free symposium. Registration is required, so please visit **http://curealz.org/symposium** to sign up. If you're unable to attend in person, you can view the symposium live at **http://curealz.org/symposium**.

ON DISPLAY: ***Love, Loss, and Laughter: Seeing Alzheimer's Differently,*** by Cathy Greenblat, Ph.D.

Tune in to our Next Free Webinar, Oct. 15

Broadcasting live from The Society for Neurosciences in New Orleans, join us on Monday, Oct. 15, at 2:30 p.m. EDT for a discussion on research progress heard at this gathering of world-class scientists. These advances will be explained and discussed by webinar moderator David Shenk, author of *The Forgetting*, with Research Consortium Chair Rudy Tanzi, Ph.D.; David Holtzman, M.D., and Scientific Advisory Board member Caleb Finch, Ph.D. To register or for more information, visit **www.curealz.org/webinar**.