

MESSAGE FROM THE CHIEF

ON OCTOBER 18, the CURE Alzheimer's Fund (curealzfund.org) awarded one of our leading researchers, Dr. Rudy Tanzi, with a \$5.4 million contribution toward our institution's mission of pursuing state-of-the-art research in uncovering the genetic roots of Alzheimer's disease. Over the course of the next 2 years, the Tanzi lab will obtain the complete genomic sequences of more than 1,500 family members that have the disease, and findings of the study will be made freely available to the global scientific communities. The CURE award represents the largest private scientific grant ever invested in whole genome sequencing, and we are immensely grateful for the trust that they and other private philanthropies have invested in us over the years. We simply could not do it without their generous support.

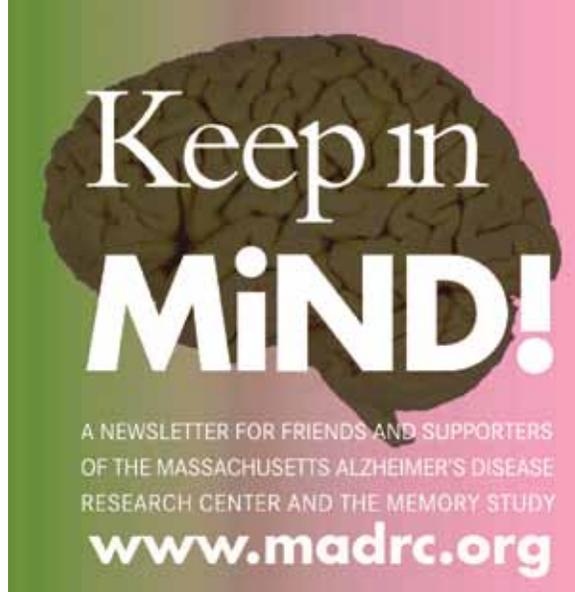
While Alzheimer's disease is the most common form of dementia, there are many other types of dementia that affect every racial and ethnic population that we know of. These include vascular dementia, dementia that occurs together with Parkinson's disease, *dementia pugilistica* (a type of dementia that affects boxers and athletes), and rapidly-progressive dementias such as Creutzfeldt-Jakob disease. We recognize the need to find effective cures for these divergent diseases, and are inspired by the recent announcement by the Nobel Prize Assembly to award the 2012 prize in physiology/medicine to Sir John Gurdon and Professor Shinya Yamanaka for their revolutionary discoveries in cellular specialization.

In this issue of our newsletter, we would like to highlight other forms of dementia that we often come across in our patients and research subjects: We have an overview of vascular dementia by one of our stroke specialists, Dr. Anand Viswanathan, a feature of the MGH Frontotemporal Dementias/Primary Progressive Aphasia Program led by Dr. Brad Dickerson, and an interesting interview with South African-born Dr. Stephen N. Gomperts, who has clinical and research interests in Parkinson's disease and dementia with Lewy bodies. Additionally, Dr. Tanzi provides our readers with an update on the 'protective gene' discovered in Iceland; a brief report of Dr. John Growdon's visit to Singapore and a September 'site-visit' tour of the MGH given to some 100 Alzheimer's Disease Centers' colleagues from across the U.S. are also included.

I hope you are inspired by and continue to support the work that we do. I wish you a most bountiful and blessed Fall/Winter season.



Brad



Dr. Hyman

Keep in MiND!

A NEWSLETTER FOR FRIENDS AND SUPPORTERS
OF THE MASSACHUSETTS ALZHEIMER'S DISEASE
RESEARCH CENTER AND THE MEMORY STUDY

www.madrc.org

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WAYS TO GIVE

For information about ways to support the clinical
care, research, teaching and community health
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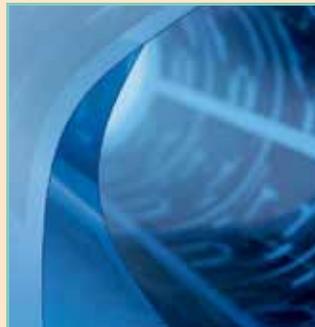
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Mixed Results from Alzheimer's Disease Clinical Trials

by Reisa A. Sperling, MD, MMSc

RESEARCHERS REPORTED the results from several large Phase 3 clinical trials in Alzheimer's disease at the American Neurological Association meeting in Boston in October. These trials tested two different antibodies (blood immune proteins), bapineuzumab and solanezumab, designed to help clear amyloid plaques out of the brain.

The bapineuzumab trials enrolled over 2,500 Alzheimer patients across the United States, divided into two studies: one trial for patients who have the most common genetic risk factor for Alzheimer's disease – apolipoprotein E e4 allele – which tested only low dose bapineuzumab, and one trial for patients without the genetic risk factor. Unfortunately, neither bapineuzumab trial showed evidence of slowing the progression of clinical symptoms in Alzheimer's disease. However, there was evidence that bapineuzumab had some effect on the biological markers tested in the study. Patients treated with bapineuzumab showed less amyloid accumulation on PET amyloid imaging and less evidence of nerve cell injury in cerebrospinal fluid tests. Although these biomarker results were somewhat encouraging, researchers think we may be trying these drugs too late in the course of Alzheimer's disease, and that we might have a better chance of slowing progression if we



started treatment at earlier stages of the disease. It may also be that we need to give higher doses of these antibodies to lower amyloid, but bapineuzumab had some side effects that limited the doses in the study.

The solanezumab trials enrolled over 2,000 patients in two studies conducted around the world. Unfortunately, these studies also did not show consistent slowing of progression of Alzheimer's disease symptoms overall but there was some evidence that the patients in the milder stages of dementia treated with solanezumab may have shown some positive effects. Looking across the two trials, the mild patients treated with solanezumab had slightly less decline in thinking and memory tests than the patients treated with placebo. Although the treatment benefits were small, researchers were encouraged that if treatments like solanezumab were started earlier in the course of Alzheimer's disease, we might be able to have a positive impact on the disease.

Researchers at the Massachusetts General Hospital and Brigham and Women's Hospital are already working on new clinical trials for Alzheimer's disease. If you or your family members are interested in participating in trials, please call 617-643-5200 for more information. ♦

New gene discovery shows protection against Alzheimer's

by Rudolph E. Tanzi, PhD and Deborah Blacker, MD, ScD

ROUGHLY 50 PERCENT of the rare early-onset familial form of Alzheimer's disease (AD) is caused by mutations in one of three genes: The amyloid precursor protein (APP) and the presenilins (PSEN1 and PSEN2). Until now, most of variants of these three genes were known to lead to AD by increasing the accumulation of amyloid beta protein (A β) in the



Flag of Iceland

brain via one of several biochemical mechanisms. Nonetheless, some in the field have remained unconvinced of the primacy of the amyloid hypothesis as the basic cause of AD. Now, compelling new evidence supporting the amyloid hypothesis

has been reported by the genetics company, DeCode, which measured the full sequence of human genes from 1,795 Icelanders. This group reported that a variant of APP seemed to be associated with a decreased risk of Alzheimer's disease: It was five times more common in cognitively normal individuals over the age of 85 than in Alzheimer disease patients. Laboratory studies of this same variant show that its biochemical actions are the opposite of those of one of the AD-causing APP gene mutations. These findings lend further support to the amyloid hypothesis -- and thus to the ongoing development of therapies aimed at lowering beta-amyloid levels in the brain as a strategy for the treatment and prevention of AD. ♦

A Conversation with Stephen N. Gomperts, MD, PhD

Dr. Gomperts, you grew up in South Africa during a particularly difficult time in the country's history. What were those years like for you? Tell us how you came half way around the world to the MGH!



Dr. Gomperts and daughter Maya

SNG: I was born in Johannesburg, in the apartheid era, but in fact my first memories date to two years that I spent in London with my family. We returned to South Africa when I was four, and there I assimilated into South African culture. I was too young to make much of the implicit inequalities that pervaded my life, but I slowly came to recognize some of them as I grew up. My parents' experience living in England drew a stark contrast with the life that they returned to in South Africa. They saw the

failings of that society through a new lens. We immigrated to California when I was seven.

I grew up in the Los Angeles area and attended UCLA, where I studied chemistry and developed a lifelong fascination with how the brain works and how it fails in disease. I therefore set off for UCSF to study neuroscience and medicine. After I acquired my MD and PhD degrees, I completed a year of internship at the Mayo Clinic, before I joined the Neurology Residency Program of the Massachusetts General Hospital and the Brigham and Women's Hospital. The experience was tremendous. After residency, I completed fellowships in Memory Disorders and Movement Disorders at MGH. During that time, I started clinical research into dementia with Lewy bodies and Parkinson's disease. I also pursued a basic research fellowship at MIT, studying the function of dopamine cells and memory cells of the hippocampus.

Did you always want to be a doctor (neurologist) and/or researcher? Who has most inspired you along the way?

SNG: I have always been fascinated with how the brain works, and as I have matured I have developed a keen interest in figuring out how to keep it working well. My father is a physician, and from an early age I considered pursuing medicine. I had the good fortune of working with an amazing and inspiring undergraduate mentor. He was a brilliant man, and he instilled in me his infectious enthusiasm for understanding the world around us. With their guidance, I honed in on a career bridging medicine and science. My PhD advisor was my next mentor. A highly talented neuroscientist, he taught me how to think rigorously and do high quality science. I have had many physician mentors, foremost Dr. John Growdon here at MGH, under whose guidance I have learned to care for patients and to hone my research questions.

I have always been fascinated with how the brain works, and as I have matured I have developed a keen interest in figuring out how to keep it working well.

You have focused your clinical and research activities on movement disorders such as Parkinson's disease and dementia with Lewy bodies. Why are you intrigued with these diseases, and how are they different from other common forms of neurologic disorders?

SNG: There are several reasons for my interest. Parkinson's disease (PD) is the second most common neurodegenerative disease, and dementia is an all too common complication. Dementia with Lewy bodies (DLB), on the other hand, is the second most common dementia. Despite their prevalence, we still have more questions than answers about these diseases. First, little is known about what causes them. Second, we need to develop tools that will let us identify and preventatively treat individuals at risk

Dementia with Lewy bodies has not received the same attention as Alzheimer's disease, but I believe that its prevalence demands it. Similarly, dementia and other nonmotor features of Parkinson's disease have only recently been subject to serious study. We have a lot of work to do.

for future disease as well as tools to treat patients once these diseases have started. Patients with DLB and PD with dementia have similar clinical problems. They share a similar pattern of cognitive impairment; and both can have motor changes known as parkinsonism, visual hallucinations, and fluctuations of thinking or alertness. Dementia with Lewy bodies has not received the same attention as Alzheimer's disease, but I believe that its prevalence demands it. Similarly, dementia and other nonmotor features of Parkinson disease have only recently been subject to serious study. We have a lot of work to do.

Protein aggregates called Lewy bodies accumulate in the brain in both of these illnesses, and in both illnesses a number of specific types of brain cells die, such as dopamine cells and cells that make the chemical acetylcholine. Their similarity suggests that any scientific insights that we can gain into one of them may be relevant to the other. Similarly, treatments that we develop for one may well work for the other.

In addition to my clinical research program focused on understanding what causes these diseases, in the lab I work to understand what dopamine cells actually do in the brain and how they do it: how they communicate with their target brain regions to guide both thinking and moving, and how that communication goes awry in PD and DLB.

How do you envision the state of research and treatment for these disorders in ten years' time?

SNG: We need to understand what causes PD and DLB, identify their risk factors, target molecular processes to prevent them, and develop drugs to treat them. It is my hope that over the next 10 years we will make great strides toward these goals. I have reason to believe that in the next decade we will develop and initiate molecular therapies that target the fundamental processes responsible for these diseases in order to achieve the above objectives. It's an exciting time to be working on these illnesses.

Lastly, tell us what you like to do during your 'down-time.' Do you think that men share similar struggles as women in achieving a better 'work-life' balance?

SNG: When I'm not working, I enjoy spending time with my wife and our toddler, Maya, who is now 1½ years old. I think that the tensions of work-life balance exist for all of us. That said, I believe that struggle is keenest for women. Although it can be a challenge to strike this balance, I count myself lucky to have the opportunity to do the work that I do and to enjoy my wonderful family.



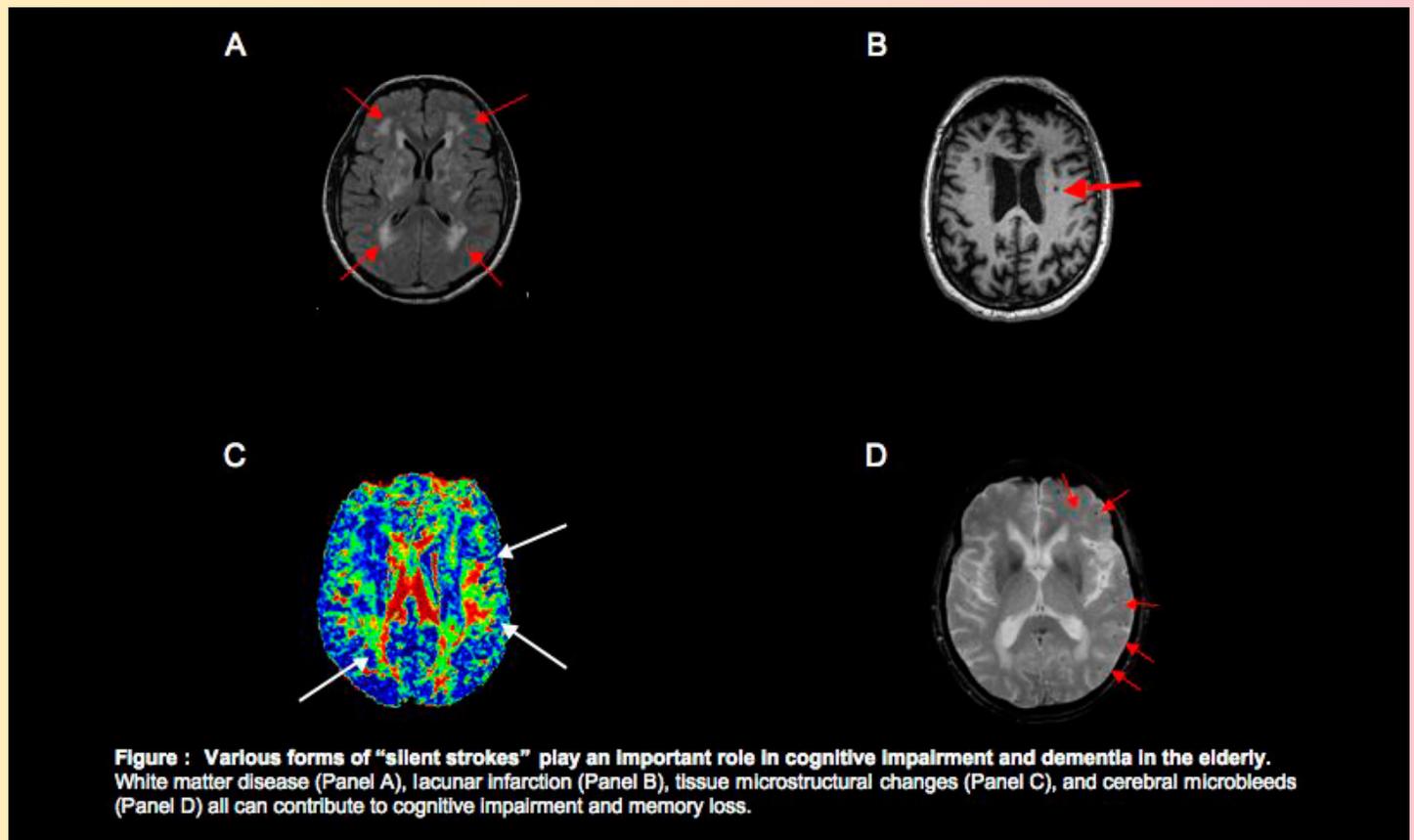
Vascular Disease and Dementia

By Anand Viswanathan, MD, PhD

IN RECENT YEARS, through research studies using advanced brain imaging techniques, it has become increasingly recognized that cardiovascular risk factors and stroke can significantly contribute to cognitive impairment in the elderly. Research from the MADRC and numerous other U.S. and international centers have demonstrated that silent strokes and brain lesions caused by vascular disease can lead to cognitive deficits and dementia. For example, the Rotterdam scan study evaluated healthy elderly individuals without stroke living in the Dutch city of Rotterdam. This study performed neuropsychological testing and brain MRI on these subjects and found that those individuals harboring a higher burden of these vascular brain lesions (or silent strokes) were more likely to have cognitive impairment or memory deficits. This study and other studies like it are in line with what has become increasingly recognized in clinical practice: vascular lesions in the brain may not be detectable in the absence of advanced brain imaging and the subtle but important influence of these lesions on cognition may only be revealed with more detailed cognitive evaluations.

Beyond traditional cardiovascular risk factors and

stroke, there exist several cerebral small vessel diseases of the brain which, through these vascular brain lesions, can cause cognitive impairment in the elderly. These diseases are independent of classic vascular risk factors such as hypertension, diabetes, or heart disease but are responsible for the development of the same vascular lesions in the brain. One such disease that appears quite common in the elderly is cerebral amyloid angiopathy (CAA). In this disease, amyloid protein (the same protein involved in Alzheimer's disease) accumulates in the small blood vessels of the brain, causing vessel fragility and dysfunction. This blood vessel dysfunction can lead to small or large areas of bleeding in the brain, cognitive impairment, or dementia. One of our ongoing studies here at the MADRC has attempted to understand the role of CAA in patients with memory impairment. We are finding that CAA is quite common in our subjects (occurring in approximately 20–30 percent of patients with memory impairment). Our ongoing goals in this study are to determine the degree of influence of CAA on cognition, how CAA affects certain areas in the brain, and how the presence of CAA can influence prognosis in patients with memory impairment. ♦

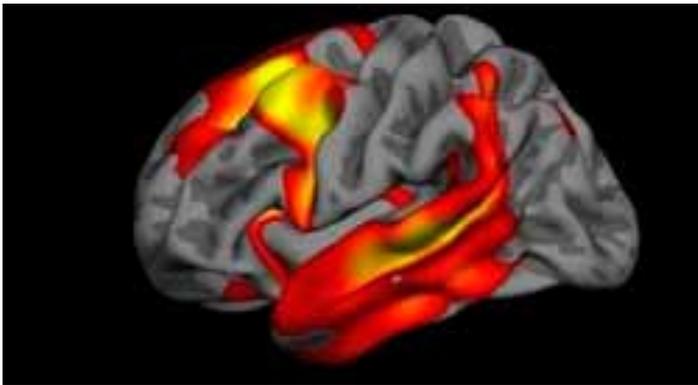


The FTD/PPA Program at MGH

by Bradford C. Dickerson, MD, MMSc, Kimiko Domoto-Reilly, MD & Daisy S. Hochberg, MS, CCC-SLP

OVERVIEW OF FTD

Frontotemporal Dementia (FTD) is a term that describes a group of brain disorders that primarily affect the frontal and temporal lobes of the brain. They are usually slowly progressive and may affect aspects of a person's social behavior, language, emotions, judgment, or other abilities. Symptoms may initially be misinterpreted as one of several psychiatric disorders, Alzheimer's disease, or even as normal aging. An accurate and early diagnosis is crucial for starting the appropriate management and planning. While there is currently no treatment to slow or reverse the damage in the brain, proactive management of the symptoms as well as support for the family may improve daily functioning for both patient and family. The primary subtypes of FTD are behavioral variant FTD, also known as Pick's disease, which causes changes in behavior, personality, and judgment, and Primary Progressive Aphasia (PPA), which involves loss of speech and/or language abilities.



Areas of the brain that are smaller in people with progressive aphasia

MGH FTD CLINICAL RESEARCH UNIT

Since its inception in 2007, the FTD Unit has benefited more than 250 patients and families with forms of FTD, progressive aphasia, and related disorders. In our clinical care of patients, we currently provide neurology, speech pathology, neuropsychology, psychiatry, social work, and genetic counseling and testing services with the main goal of maximizing and maintaining quality of life. In our research, the goals are to contribute to the understanding of FTD with the ultimate goal of developing tools to better evaluate whether new treatments are working. Some of the ways we are doing this include the following lines of research:

PROGRESSIVE APHASIA SEVERITY SCALE (PASS):

We developed the PASS as a scale to rate the presence and severity of difficulties in a variety of aspects of speech,

language, and functional communication skills. The goal is to provide health care providers and investigators with a tool that can be used in identifying strengths and weaknesses in a patient's communication skills which can assist in diagnosis and treatment planning, as well as monitoring symptom progression over time. We have taught a number of other teams around the world how to use it and are hoping it will be incorporated into studies of new treatments.

SOCIAL IMPAIRMENT RATING SCALE (SIRS):

Similarly, we have just finished developing a new scale to rate the presence and severity of difficulties in a variety of aspects of social behavior, again with the goal of developing an instrument for use in research and treatment development. One obstacle in the past has been that such instruments have not existed, and therefore it has been difficult to measure whether treatments are improving these specific kinds of abilities.

NEUROIMAGING AND FLUID BIOMARKERS:

We have worked for many years on the development of advanced neuroimaging (particularly MRI and PET) methods to measure abnormalities in the structure, function, and molecular makeup of the brain. We are just completing an initial phase of work to translate these methods into a standardized measure called the "FTD signature," which measures the size of brain regions typically affected in FTD and we hope will be useful for early diagnosis and monitoring progression and treatment development. In addition, we have been working on measurements of genes and proteins in blood and spinal fluid for similar purposes. Building on our initial work, we are now participating in two multi-center studies on these topics, the "Neuroimaging in Frontotemporal Degeneration (NIFD)" and the "4-repeat Tau Neuroimaging Initiative (4rTNI)," focusing on two related conditions (Progressive Supranuclear Palsy and Corticobasal Degeneration).

FTD FAMILY AND GENETIC STUDIES:

In some families, FTD is inherited from one generation to the next. We are enrolling as many members of such families as possible in our ongoing study so that we can collect blood samples and follow individuals over time to learn about the cognitive abilities, personality, and other traits of people who may be at risk for this condition but do not have symptoms. We hope the knowledge gained from this work will help researchers to learn if there are certain cognitive or behavioral traits that can help predict the development of FTD. ♦

Some Thoughts from Our Participants

MANY YEARS AGO, at a family reunion, a relative that I hadn't seen in a while was not the "same" person I remembered her to be. I bring this up because she exhibited similar traits that my mother had when she was diagnosed with dementia many years later. The point being that my mom's illness progression was so subtle that I thought this was part of the aging process as mom was in her 80's at the time. In both instances I had no idea what was happening.

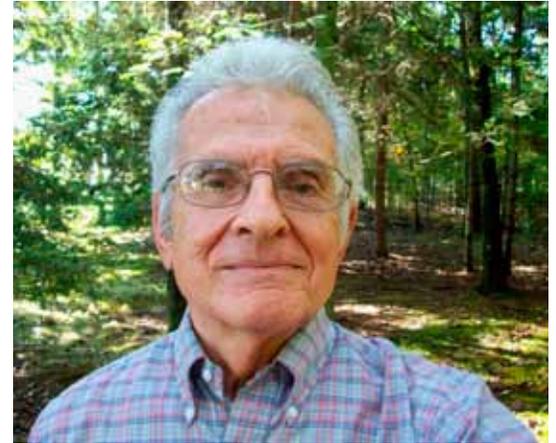
Toward the end of my 45 years in engineering, I began to notice that it was becoming more of an effort to retain data that, for so long before, was not an issue. I was the go-to guy for information on past projects and the like. I surmised that my brain's "hard drive" was nearing capacity or was this the first subtle indication of something else, which eventually led to my entering the brain study program.

Being part of the study has been primarily an educational experience. Knowing how aging normally affects one's brain verses non-aging issues has eased my concerns. This particular aspect is an important component to me and I will endeavor to learn more about the brain as time passes.

Participating in this program has been very rewarding, knowing in some small way I'm contributing to the knowledge base of this very important study. The best part is the continued support from my family, meeting and interacting with the study professionals. Thank you all!

Alfred DiBonaventura

North Grosvenordale, CT, October 5, 2012



Alfred DiBonaventura

MY SPEECH started deteriorating about ten years ago and I was having a hard time getting a definitive diagnosis; so I moved back home to Boston since I knew I would find the greatest range of diagnostic and treatment resources in my adopted city. I was quickly introduced to the doctors and programs at the MassGeneral FTD Unit, ADRC, and MassGeneral Institute for Neurodegenerative Disease (MIND). As a result, I got both a mission; and along the way, the long elusive identification of my condition.

Primary progressive apraxia of speech has no treatment yet; but the flip side of that coin is that the neurologists and health professionals at the FTD Unit, ADRC, and MIND are aggressively pursuing research on the causes of related neurodegenerative conditions. Participating in their longitudinal studies is a perfect fit for a person who has always been deeply interested in science, and I'm delighted to know my involvement will likely help the people who are affected by neurodegenerative diseases in any form going forward.

Volunteering in the research studies also satisfies my intellectual curiosity about apraxia; and a special bonus is that I get to be an early adopter participant in the latest medical toys like the three-dimensional PET/MRI, the first combination imaging scanner in this country. Better living with technology!

Paying it forward for the exceptional care I've been shown by the dedicated professionals at MIND is more like winning the lottery: I'm largely the beneficiary.

The opportunity to participate in preeminent research, collaborate with leading professionals in their fields on my health care management, and being inspired to continue to contribute to research outcomes after I die by donating my brain are chances I would not have missed for the world. Finally, I'm going to Harvard – or at least my brain is! Thank you for the great experience.

Betsy Fedak Gethin

Watertown, MA, October 4, 2012



Betsy Fedak Gethin

Greetings from the Clinical Coordinator

To all our Memory Study participants

Fall is here and we're busier than ever in the Gerontology Research Unit. We're continuing to work hard to understand more and more about normal aging and the various neurodegenerative diseases.

We're asking you to continue to do for us what you've done year in and year out ... and, for some of you, we're asking you to do some new tasks, too. There are additional forms that you fill out that give us more information about how you are doing in your daily activities. Some study participants have done novel paper-and-pencil tests. You've helped us understand more about biomarkers by giving us blood samples and cerebrospinal fluid. Some of you have participated in affiliated studies that involve various neuroimaging techniques. Many have done the "smell test" and we've just begun to administer an eye-tracking test. And many of you have expressed interest in brain donation and have had discussions with your families about this. This is a very important tool in helping us learn how the brain changes in healthy aging and in disease.

Sometimes you may wonder why in the world we're asking you to do all this! The brain is a very complicated organ. In addition to changes that occur as one ages normally, there are numerous diseases that can afflict the brain. We know much about the brain but there's a lot more to learn. Doctors in the field have theories and ideas. But their theories are only as good as the data behind them. And that's where you come in! You are the fuel that drives the research. So thank you again for your time and patience and perseverance! We couldn't do this work without you!

Jeanette Gunther
Clinical Coordinator

Announcing A New Centralized Telephone Number For Inquiries About Our Studies!

Interested in learning more about research studies and how you can get involved? Patients, caregivers, family members, and healthy volunteers can call **Caroline Sullivan at 617-643-5200** to learn about all of the exciting research opportunities we have going on at the MADRC! ♦



Alzheimer's Association Annual Greater Boston Walk to End Alzheimer's

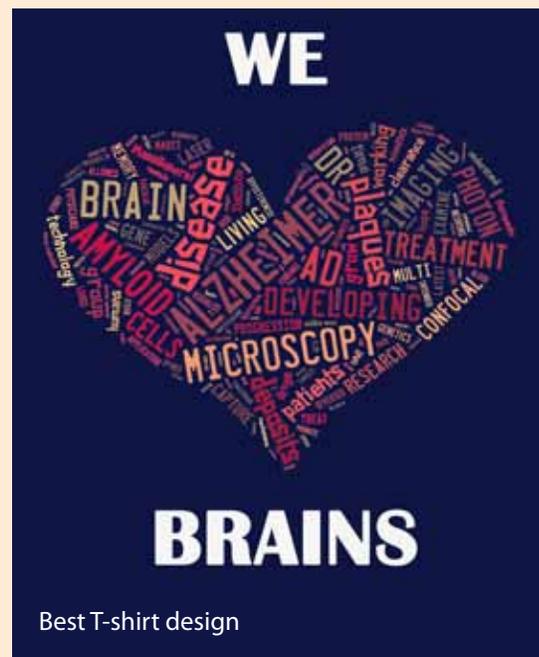
By Naomi B. Shakerdge, BA, Team Captain

ON SUNDAY, SEPTEMBER 23RD, 70 participants came together to represent the Massachusetts Alzheimer's Disease Research Center at the Alzheimer's Association's annual Greater Boston Walk to End Alzheimer's. As our team made its way to the starting point of the walk, we were greeted by various vendors and had the opportunity to take part in a number of fun activities such as dancing and an aerobics class. All participants had the chance to place their colorful windmill flowers in the "Promise Garden" – each color representing one's relationship to Alzheimer's (i.e. green symbolizing being a caregiver for someone with Alzheimer's).

For the second year in a row, I had the privilege to lead our research center in this walk. This year we raised \$4,230. Along with the help of Hyman lab members, Muriel Arimon and Ashley Winslow, we designed our team t-shirt that was awarded "Best T-shirt Design" by the Alzheimer's Association. The front of our t-shirt was titled "We Heart Brains" with the heart encompassing a variety of phrases related to Alzheimer's disease, such as "amyloid" and "tangles." The back of the t-shirt featured a neuron with the caption "Let's keep them healthy." As our team marched off, I felt such a strong sense of community among the hundreds of teams, all dedicated to the same cause. ♦



The cool crew of the Hyman Lab team



Best T-shirt design



The Promise Garden display

CONGRATULATIONS TO DR. BRAD HYMAN ...

the Director of our Alzheimer's Disease Research Center, for receiving the 2012 Henry Wisniewski Lifetime Achievement Award from the Alzheimer's Association at its International Conference held in Vancouver, Canada during the summer!!

More information about the award and Dr. Hyman's work may be found at www.alz.org/aaic/about/award-winners.asp



A 'site visit' to welcome our nationwide colleagues

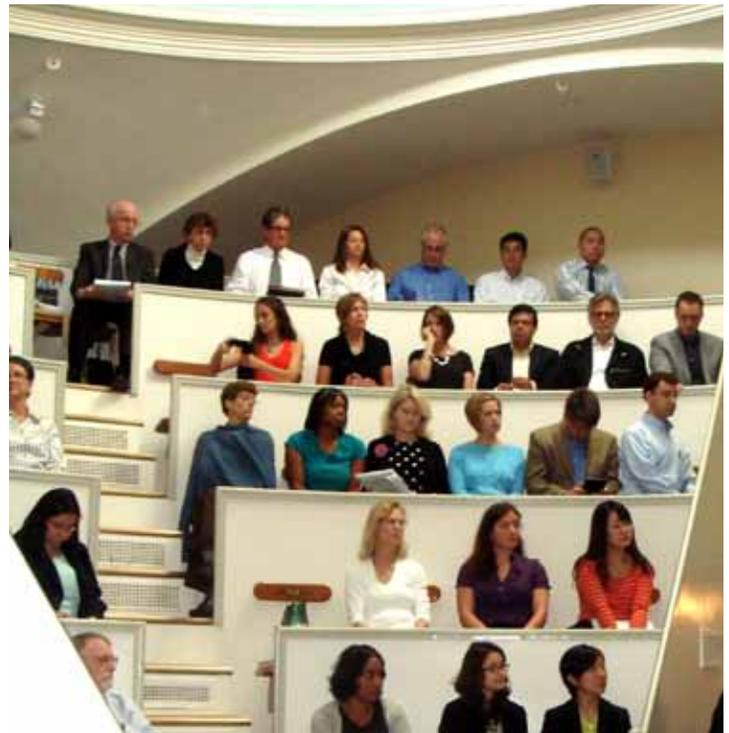
by Liang Yap, PhD

WE INVITED approximately 100 friends and colleagues from the National Institute on Aging (NIA) and all Alzheimer's Disease Centers (ADCs) to a tour of the MGH on October 5, 2012. They were in town to attend the annual Fall ADCs meeting, which was held in conjunction with the American Neurological Association's (ANA) meeting in Boston.

Visitors were treated to breakfast and a morning mini-symposium on the 'Detection and Intervention in Preclinical Alzheimer's Disease' in the historic MGH Ether Dome, with presentations from Drs. Keith Johnson, Teresa Gomez-Isla, Dorene Rentz and Reisa Sperling. They then travelled to the MassGeneral Institute for Neurodegenerative Disease (MIND) in Charlestown, where they attended a choice of several small 'breakout sessions' – with topics ranging from neuroimaging, new assessment methods to detect/monitor preclinical Alzheimer's disease, data management, to venture philanthropy and research.

At MIND, our friends and colleagues also toured our research laboratories and were treated to a delicious array of lunch items catered from the Milk Street Café. Each visitor left with a folder filled with 'goodies' – copies of our popular newsletters, MGH notebooks and pens, and the following brochures: 'MGH History Trail: A Walking Tour of the MGH', 'The Birth of Modern Surgery/The Ether Dome', 'Dr. Paul S. Russell Museum of Medical History and Innovation', and 'Building 114: MGH's Newest State-of-the-Art Biomedical Research Laboratory'.

It seems that everyone enjoyed the much-anticipated 'site-visit', and we were delighted to have played a small role as 'ambassadors' of our historic city! ♦



Attentive audience in the Ether Dome.
Left: Dr. Rudy Tanzi

Keep in MiND!

A NEWSLETTER FOR FRIENDS AND SUPPORTERS
OF THE MASSACHUSETTS ALZHEIMER'S DISEASE
RESEARCH CENTER AND THE MEMORY STUDY

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Dr. John Growdon at the Singapore Shangri-La Hotel luncheon on May 9, 2012



Keynote speaker at the Singapore 2012 Annual Conference on Aging

Presence at the Annual Singapore Conference on Aging (ASCA) 2012

by Liang Yap, PhD

DR. JOHN GROWDON was invited to be the keynote speaker at ASCA on May 10, and presented the 5th Henry Lim Lecture on 'The Heart-Brain Partnership: The Good, The Bad, and The Complex' – an overview of the medical and scientific findings on the relationship between cardiovascular disease and cognitive impairments. The topic was of immense interest to the predominantly Asian participants of the conference who had hailed from the academic, service and health-care sectors – including Singapore's Minister of Health, who was the guest-of-honor.

Ischemic heart disease and cerebrovascular disease (including strokes) are principal causes of death amongst a variety of Asian ethnic groups, and the prevention and treatment of these medical conditions are the major focus of a large number of research institutions throughout Asia.

Besides attending the ASCA, Dr. Growdon's lecture circuit also included the National University of Singapore/National University Hospital, and a private event for key policy-makers. ♦