My first memory of Alzheimer’s disease (AD) is quite poignant and left an indelible mark in my mind as a child. We lived in California, and our family traveled to Iowa for a family reunion. The main hallmark of this event was to visit my maternal great-grandmother for the first and last time. I had never met her, but I remember my mother looking forward to seeing her grandmother, whom she had fond memories of. Mind you, this was not a visit, but a last farewell to a woman who meant a great deal to the family. I will never forget seeing her tied to her wheelchair, unable to sit up straight, unable to communicate, and with no hint of recognition in her empty eyes. This was a great deal for a ten year-old to process.

The Genetics of Alzheimer’s Disease and Related Dementias

Scientists don’t yet fully understand what causes Alzheimer’s. However, the more they learn about this disease, the more they realize that genes play an important role in its development. Some diseases are caused by a genetic mutation or permanent change in one or more specific genes. If a person inherits from a parent a genetic mutation that causes a certain disease, then he or she will often get the disease. In other diseases, a genetic variant may occur. This change in a gene can sometimes cause a disease directly, but more often, it acts to increase or decrease a person’s risk of developing a disease or condition. When a genetic variant increases disease risk but does not directly cause a disease, it is called a genetic risk factor.
The Journey From Genetic Testing to Generating Hope

[CONTINUED FROM COVER PAGE]

Many years went by. Our family lived our lives without any more concern of AD (called dementia at the time) when AD reared its insidious head once again. My grandmother was diagnosed with AD in her 70s along with two of her brothers, my great uncles. To watch my mother care for my grandmother was heart wrenching to say the least, but I never quite equated AD with genetics. So when I volunteered to participate in a study to see whether persons would change their lifestyles if they knew they were at a genetic risk for certain diseases, AD was not even on my radar screen.

My motive for participating in the study was to find out what my risk for another disease was, as I had experienced some neurological symptoms. At this point in time, my father was now living with AD, and I should not have been surprised to have been informed that I have two copies of the ApoE 4 allele which puts me at a 91% life time risk of having AD. I was in “healthy denial” about AD at the time, and I will never have that tool to use again as I watched my father decline and eventually pass with AD. The sad thing is that I was not prepared to know this information. I can honestly say that if I had been educated on what I was being tested for and what the results would mean in the way of risk, I would NOT have chosen to be tested. There is no prevention or cure for AD at this point in time, and I felt very lonely and traumatized by knowing my genotype. All of a sudden my world had changed and I lost hope and direction. My days were filled with fear, anxiety, and sadness. I knew of no one who had this genotype and felt very lonely. I mean, what does one do with this information?

Then, the moment of truth arrived. I could no longer live like this and it had become quite disruptive to me and my family. I decided to reach out and get help. It was then that I was diagnosed with post traumatic stress disorder (PTSD) and began to embark on a new path. I soon realized that life is a journey, things happen for a reason, and the information was not to be feared but conquered! The loss of my life’s vision was now becoming a distant enemy and the road to making a difference began! My whole life of being a nurse and having a career in business development, my family history of AD, and my knowledge of my own possible future had prepared me for this moment. I have been blessed with a purpose and now it is time to run with it!

Upon reflecting and experiencing great gratitude from my girlfriends, and realizing that women are most affected by AD due to caregiving responsibilities, B.A.B.E.S., “Beating Alzheimer’s by Embracing Science” was born. We are a non-profit charity and our mission is to raise funds to be directly donated to the most promising research projects to help prevent or find a cure for AD. There is only one way out of this disease, and it is through research. There is so much to be done, so many funds to be raised, and so many lives to change. Making a difference is the most healing thing that can be done in one’s recovery from a traumatic event, and I feel blessed to be here today. UC San Diego’s Shiley-Marcos Alzheimer’s Disease Research Center (ADRC) has been very kind in helping with this process, and I am forever grateful.

Please consider giving a donation to B.A.B.E.S. or directly to UC San Diego Shiley-Marcos ADRC. Together we can make a difference!

For more information: contact jamietyrone@cox.net or visit http://www.AlzBabes.org
Alzheimer’s Disease Advocacy Update
BY MARY SUNDSMO, MBA

In April, I represented the San Diego/Imperial Chapter of the Alzheimer’s Association as a delegate to the 2012 Advocacy Forum in Washington DC. Although the Shiley-Marcos ADRC is an entirely separate entity from the Alzheimer’s Association, I have done volunteer advocacy work with the Association for many years. I have been making this trip to Washington each spring for more than ten years.

The air was charged with excitement at the 2012 Advocacy Forum. In January, 2011, President Obama signed into law The National Alzheimer’s Project Act (NAPA), the largest legislative victory for Alzheimer’s in over 18 years. NAPA required a national plan for Alzheimer’s disease (which was released in May, 2012 after our advocacy visits). Secretary of Health and Human Services, Kathleen Sibelius, was coming to talk to the 650 advocates at the Forum about implementing the National Plan. The Forum is designed to have two days of training and then one day “on the Hill” visiting our local Congressmen, putting into words the things we learned in training. As the sole representative from San Diego, I had my plate full with five Congressional visits: Members Davis, Filner, Hunter, Issa, and Bilbray.

Sometimes we meet the Members themselves, but more often with Congressional staffers, some of whom are just new to their jobs. Others may be more senior legislative analysts specializing in health matters. Advocates are trained to make ‘asks’ that are quite specific. We had two ‘asks’ this year.

1) Support the National Alzheimer’s Plan with the resources needed to change the trajectory of AD.
NAPA did not address financing. In January 2012, the President allocated $100 million for AD research, education, outreach, and support activities in his FY 2013 budget. We asked that these monies be supported through the Congressional appropriation process.

2) Support the HOPE [Health Outcomes, Planning, and Education] for Alzheimer’s Act.
This bill asks for funding to require certain items be included in a doctor’s visit for Medicare reimbursement to physicians: documentation of formal diagnosis in an electronic medical record and access to information, resources, and services through care planning for families.

My favorite training session was given by Frank Luntz, a well-known political commentator. His mission was to give us ‘words that work’ on Capitol Hill. Our challenge is to be memorable to these Congressmen and their staff since they have so many people visiting each day asking for support. A few statements he made really stuck with me. I used these comments in my visits on the Hill.

“If Congress does not fund research, eradicating the disease won’t happen. Then stand up to our families and say why.”

“If you remember only one thing from our conversation, remember this: The epidemic of AD will only get worse.”

I felt that our issues were heard more than ever. Many were aware of the Alzheimer’s National Plan and the HOPE Act. They listened and took notes, commenting that they would relay the information to their Congressman. In an election year, it is very difficult to get anything through the budget process. So while I felt like I was convincing in my arguments in support of our “asks”, I was also realistic knowing that the chances for funding were also slim.

Awareness about AD has grown greatly in the last few years. This really helps our advocacy efforts as constituents can pressure their elected officials to support some of these Alzheimer’s related bills. If you are interested in raising your voice with your elected officials, please contact me at the Shiley-Marcos ADRC and I can give you some ideas of how you might help.
There are two variants of Alzheimer’s disease (AD)—early-onset and late-onset. Both have genetic components.

**Early-Onset AD**

Early-onset AD occurs in people age 30 to 60. It is rare, representing less than five percent of all people who have AD. Some cases of early-onset AD have no known cause, but most are inherited, a type known as familial AD (FAD).

Familial AD is caused by any one of a number of different mutations in specific genes on chromosomes 21, 14, and 1. Each mutation causes an abnormal protein to be formed. The abnormal proteins are amyloid precursor protein (APP) (located on chromosome 21), and presenilin 1 and 2 (on chromosomes 14 and 1, respectively).

Each of these mutations plays a role in the breakdown of APP, a protein whose precise function is not yet known. This breakdown can generate harmful forms of amyloid beta protein, which may clump and form plaques, a hallmark of AD. A child whose mother or father carries a genetic mutation for FAD has a 50/50 chance of inheriting that mutation. Someone who inherits one of these mutations almost surely will develop FAD, often with a similar age of onset as the parent.

Research about early-onset AD has helped identify key steps in the formation of amyloid and plaques, some of which are targets for treatment.

National Institute on Aging-supported scientists are continuing this research through the Dominantly Inherited Alzheimer Network (DIAN), an international partnership to study families with genetic mutations that cause early-onset AD. By studying changes that occur in these families long before symptoms appear, scientists hope to gain insight into mechanisms of early- and late-onset AD. Novel forms of treatment will also be studied in the DIAN cohort.

**Late-Onset AD (LOAD)**

Most cases of AD develop after age 60. The risk of late-onset AD (LOAD) may be influenced by genetic, environmental, and lifestyle factors.

Several genetic risk factors may predispose to LOAD. The strongest is the apolipoprotein E (APOE) gene found on chromosome 19, which codes for a protein that helps transport cholesterol and other lipids. APOE comes in three major forms (or alleles) e2, e3, and e4.

- APOE e2 is relatively rare and may decrease the risk of LOAD.
- APOE e3, the most common allele, neither decreases or increases risk.
- APOE e4 is present in about 25 to 30 percent of people and in over 40 percent of people with LOAD. Therefore APOE e4 increases the risk of developing Alzheimer’s.

How the APOE e4 allele increases the risk of developing AD, is not fully understood. Variation in the APOE gene helps to explain some of the variation in the age at which AD may develop. Inheriting an APOE e4 allele does not mean that a person will definitely develop Alzheimer’s. Some people with one or two APOE e4 alleles never get the disease, and others who develop Alzheimer’s do not have any APOE e4 alleles.

Using a relatively new approach called genome-wide association study
(GWAS), researchers have identified a number of genes in addition to APOE e4 that may increase a person’s risk for LOAD, including BIN1, CLU, PICALM, and CR1. Identifying genetic risk factors helps scientists better understand mechanisms of AD.

**Genetic Testing**

For people at risk of early onset AD, testing the Presenilin and APP genes for mutations may provide definitive information about ultimately developing the disease. Genetic counseling prior to testing is recommended.

For LOAD, the implications of genetic testing are less clear. Although a DNA test can identify someone’s APOE alleles, it cannot predict with certainty whether or when they may develop AD. At present, APOE testing is used in research settings to identify study participants who may have an increased risk of developing AD. This knowledge helps scientists look for early brain changes in participants and compare the effectiveness of treatments for people with different APOE profiles. Most researchers believe that APOE testing is useful for studying AD risk in large groups of people but not for determining any one person’s specific risk.

In doctors’ offices and clinical settings, genetic testing is used for people with a family history of early-onset AD. However, it is not generally recommended for people at risk of late-onset AD.

**Major AD Genetics Research Efforts**

The National Institute on Aging supports several major genetics research programs:

- The Alzheimer’s Disease Genetics Study is gathering and analyzing genetic and other information from 1,000 or more families in the United States with two or more members who have late-onset Alzheimer’s.
- The Alzheimer’s Disease Genetics Consortium is a collaborative effort of geneticists to collect and conduct GWAS from thousands of families around the world with members who do and do not have late-onset AD.
- The Dominantly Inherited Alzheimer Network (DIAN) is an international research partnership studying early-onset familial AD in biological adult children of a parent with a mutated gene.
- The National Cell Repository for Alzheimer’s Disease (NCRAD) is a national resource where clinical information and DNA samples are stored and made available for analysis by qualified researchers.

The participation of volunteers is a critical part of AD genetics research. The more genetic information that researchers can gather and analyze from a wide range of individuals and families, the more clues they will have for finding additional risk-factor genes.

To learn more about participating in Genetic studies, contact NCRAD at 1 (800) 526-2839 or www.ncrad.org

This article is adapted with permission from the National Institutes of Health Publication Alzheimer’s Disease Genetics, No 11-6424, June, 2011

**Genes involved in Frontotemporal and Lewy body dementias**

**Frontotemporal dementias (FTD):**

Frontotemporal dementia (FTD) is less common than AD, although it may account for 50% of people with onset of dementia before age 60. FTD consists of a number of disorders, some with behavioral changes, others with progressive language impairment. An increasing number of genes have been linked to FTD in recent years. Mutations in these genes almost always predict developing the disease.

- **Tau gene** (also called the MAPT gene, on chromosome 17)—mutations in this gene affect either the structure or levels of tau protein, leading to clumping of tau and damage to nerve cells (neurons).
- **Progranulin (PGRN) gene**—mutations in PGRN lead to lower levels of the protein progranulin, which somehow alters the function of another protein, TDP-43. People with PGRN mutations may develop familial forms of FTD or primary progressive aphasia.
- **C9orf72 gene**—An unusual alteration in this gene, first identified in 2011, appears to be the most common genetic cause of familial frontotemporal disorders (about 25%) and has been found in non-familial FTD (6%). This mutation also occurs in ALS (Lou Gehrig’s disease).
- **VCP gene and CHMP2B gene**—Rare mutations in these genes lead to familial FTD.
- **Mutations in two other genes, TDP43 and FUS** are associated with familial ALS, but rarely, FTD.

Genetic discoveries in FTD have provided better ways to identify mechanisms of disease and develop therapies.

**Lewy Body Dementias:**

Lewy Body dementias include Parkinson’s Disease Dementia (PDD) and Dementia with Lewy Bodies (DLB). These share the pathology of abnormal forms of a protein called alpha-synuclein (ASYN) in the brain. Although mutations in at least ten genes predisposing to Parkinson’s disease have been identified, these are less clearly related to typical late-onset PDD or DLB. Genetic testing is therefore not recommended for people with these types of disorders with onset in later life.
Knowledge of APOE Genotype Affects Subjective and Objective Memory Performance in Healthy Older Adults

BY DAVID SALMON, PhD

There have been exciting advances over the past few years in our ability to detect biological markers that signal an increased risk of developing Alzheimer’s disease (AD) and dementia. One of the best known of these markers is a gene located on chromosome 19 that is involved in the production of a protein called apolipoprotein E (APOE). If a person has a form of the APOE gene made up of one ε4 allele (each gene is made up of two alleles), they have double the chance of developing Alzheimer’s disease as they age. If a person has an APOE gene with two ε4 alleles, they have eleven times the chance of developing AD.

Recent research and debate has focused on the risks, benefits, and general ethics of disclosing APOE genotype to older adults. A study that was published in 2009 in the New England Journal of Medicine suggests that disclosure has few adverse emotional risks. Groups of healthy older adults who were randomly assigned to a disclosure group informed of their APOE gene status or a non-disclosure group who did not receive this information did not differ in levels of depression or anxiety during the year following disclosure. This was true regardless of whether disclosure revealed ε4+ or ε4- gene status.

A question that has not been addressed, however, is how knowledge of one’s APOE genotype might affect self-judgment of memory functioning. Because the devastating impact of AD on the ability to remember is widely known, older adults who know they have a genetic risk for the disease might have lower confidence in their memory and be more likely to think their everyday memory is worse than those who do not have the risk or do not know their genotype. It is also possible that knowledge of genetic risk for AD could influence objective memory performance. Knowledge that you have a characteristic associated with poor memory performance could lead to underperformance on objective memory tests due to low confidence, reduced effort, or lack of perseverance.

To examine these possibilities, we conducted a study in which we tested memory performance and obtained memory ratings from cognitively normal elderly individuals with known APOE genotype who were either informed (with appropriate genetic counseling) or not informed of their genotype prior to memory evaluation. We hypothesized that older adults with knowledge of their ε4+ status would judge their memory more harshly and have worse objective memory test performance than those without knowledge of their ε4+ status. In contrast, older adults with knowledge of their ε4- status would judge their memory more positively and have better objective memory test performance than those without knowledge of their ε4- status.

The study included 50 healthy elderly adults who were APOE ε4+ and 94 who were APOE ε4-. Approximately half of the APOE ε4+ and ε4- participants knew their gene status at the time memory was evaluated. The objective memory evaluation and memory ratings followed genotype disclosure by an average of about eight months. The memory tests included a verbal test that required people to recall two brief stories that are read aloud to them, and a visual test in which they copied a complex abstract line drawing as precisely as possible, then reproduced the figure from memory immediately or after a 30-minute delay. Memory self-ratings were made with two scales. The first was the Metamemory in Adulthood Questionnaire that asked participants to rate 15 of their current everyday memory abilities on a 5-point scale. The second was the Memory Functioning Questionnaire on which participants rated 46 items about their memory on a 7-point scale. The items included questions about current compared to past memory functioning, how often problems arise in specific memory situ-
Informing older adults that they have an APOE genotype associated with increased risk of AD can have adverse consequences on their perception of their memory abilities and performance on objective memory tests.

The results of our study showed that older adults with knowledge of their APOE ε4+ status tended to judge their memory more harshly and performed worse on an objective verbal memory test than did ε4+ adults without that knowledge. On the other hand, older adults with knowledge of their APOE ε4- status judged their memory more positively than did those without knowledge that they were ε4-, and the two ε4- groups did not differ significantly in objective memory test performance (see figure). The different patterns of results we observed in the ε4+ and ε4- groups could not be attributed to differences in age, level of education, or gender since the groups did not differ on these characteristics. Importantly, there was no evidence of depression or increased anxiety in any of the groups.

Why was objective memory test performance adversely affected by knowledge that one has a risk factor for AD? One possibility is that ε4+ older adults who knew they were at risk for AD had lowered expectations that resulted in reduced persistence and effort in performing memory tasks. On the other hand, high expectations regarding their memory abilities may have led ε4+ older adults who knew they did not have the risk factor to apply more effort to test performance. These possibilities are consistent with the pattern of subjective memory ratings we observed, and with the fact that memory self-ratings were significantly correlated with objective memory test scores across all groups. Further research is needed to verify these possible explanations.

Our study has several limitations that should be considered. First, we did not randomly assign participants to disclosure or non-disclosure groups, so strong inferences about cause and effect relationships cannot be made. Second, the study is cross-sectional so the long-term impact of knowledge of APOE genotype on memory ratings and objective memory performance cannot be determined. Despite these limitations, our results indicate that informing older adults that they have an APOE genotype associated with increased risk of AD can have adverse consequences on their perception of their memory abilities and performance on objective memory tests. Similar consequences might be expected if other biomarkers for AD risk are disclosed (e.g., neuroimaging or cerebrospinal fluid biomarkers of preclinical AD). This could have a serious clinical impact by increasing the likelihood of an incorrect diagnosis of dementia or Mild Cognitive Impairment in those who know they are APOE ε4+, and it could distort the results of AD prevention trials if healthy elderly with knowledge of their APOE ε4+ status are over-represented in clinical drug trials. It is clear from our results that clinicians and researchers should consider patients’ knowledge of their genotype and possession of other AD biomarkers when evaluating older adults who may or may not be at risk for developing dementia.
Helpful Resources

Home Safety for People with Alzheimer’s Disease

This 40-page booklet was originally written by staff at the Shiley-Marcos ADRC. The booklet was revised and updated by Alzheimer’s Disease Education and Referral (ADEAR) and is now available through this helpful organization.

Home Safety for People with Alzheimer’s Disease gives room-by-room suggestions for maximizing safety for people with Alzheimer’s or a related dementia. The publication also addresses common behavioral challenges related to dementia and how to manage them through environmental adaptations or adjustments. The topics of driving as well as planning for a natural disaster are addressed in the booklet as well as the special consideration of people who live alone. Helpful Checklists and additional resources make this a very practical guide.

You can order a free print copy of the booklet through ADEAR by calling 1-800-438-4380. You can also order the booklet or download the PDF at:

Simple Solutions: Practical Ideas and Products to Enhance Independent Living

Many people hope that as they age or develop dementia, they will be able to stay in their own home as long as possible. With the advent of more technology and some innovative planning, there are many ways in which homes or day-to-day activities can be modified to support greater safety and independence.

The Hartford offers a variety of excellent publications free of charge. “Simple Solutions – Practical Ideas and Products to Enhance Independent Living” includes descriptions of over 200 products and ideas that can enhance independence, safety, and comfort for seniors in their homes, including those living with dementia.

Contents include:
• Vision Solutions
• Hearing Solutions
• Mobility and Balance Solutions
• Strength, Dexterity, and Reach
• Memory Solutions
• Fire and Burn Safety Solutions

The 52-page booklet includes a supplier guide that references where each suggested item can be purchased, as well as a home checklist to enable the reader to assess the safety and convenience of his or her own home.

Modern Ideas, Modern Living: Taking the Next Step in Home Design and Planning for the Lifestyle You Want

Some individuals or couples living with dementia face decisions about whether to move to be closer to family or services; whether to downsize or settle into a retirement community; or whether design modifications to an existing home can make it more livable and accommodating to both physical and cognitive aging.

Modern Ideas, Modern Living is a 32-page guide that provides helpful steps and questions, as well as priorities to consider when making these important decisions. It includes a number of “universal design” principles and checklists for homes that make them safer and more livable.

These Hartford publications are available to download in PDF format free of charge. Those residing in North America can also order print copies delivered by mail.

To access or order these Hartford booklets and other publications, see:
http://www.thehartford.com/life

Click onto resources to find these publications and other resources.
# Clinical Trials

## Alzheimer’s Disease Neuroimaging Initiative 2 (ADNI 2)

**Principal Investigator:** James Brewer, MD, PhD  
**Time Involved:** 4 Years | **Contact:** Helen Vanderswag, RN - (858) 822-4800

The purpose of the study is to examine how brain imaging technology and biomarker tests, along with measurements of memory and daily functioning, can be used in the future conduct of studies that focus on the identification and treatment of AD at an early stage.

**Requirements:**  
- Early memory problems, a diagnosis of MCI or AD, and those without memory changes  
- 55-90 years old; 65-90 for normal controls  
- Have a study partner for all visits  
- Able and willing to undergo MRI, PET scans, and lumbar puncture procedure (LP)  
- MMSE score of 20 or above

## Roche WN25203B (SCarlet RoAD)

**Principal Investigator:** Michael Rafii, MD, PhD; Judith Rivera, NP  
**Time Involved:** 24 Months | **Contact:** Kacie Smith - (858) 246-1303

Randomized, double-blind, placebo-controlled, parallel-group two-year study to evaluate the effect on cognition and function in prodromal Alzheimer’s disease of subcutaneous gantenerumab.

**Requirements:**  
- 50-85 years old  
- Prodromal AD with MMSE greater than 24  
- Have a study partner for all visits  
- On no memory medications

## Resveratrol (ADC-037-RES)

**Principal Investigator:** Michael Rafii, MD, PhD; Judith Rivera, NP  
**Time Involved:** 12 Months | **Contact:** Jennifer Foster - (858) 246-1306

Phase 2, double-blind, placebo-controlled, parallel arm drug trial to evaluate the safety, tolerability, and effectiveness of resveratrol when given to people with mild-to-moderate AD. All participants will undergo CSF collection (lumbar puncture) and volumetric MRI.

**Requirements:**  
- Age 50 or older with mild-to-moderate AD  
- MMSE score 14-26 (inclusive)  
- Have a study partner for all visits  
- Able to abstain from eating large quantities of resveratrol containing foods or resveratrol dietary supplements

## Nerve Growth Factor

**Principal Investigator:** Michael Rafii, MD, PhD  
**Time Involved:** 24 Months | **Contact:** Michelle Herman - (858) 246-1303

Nerve Growth Factor (NGF) research is a phase 2 double-blind placebo controlled study to test the safety, tolerability, and effectiveness of a gene transfer drug called Cere-110 in those with mild-to-moderate AD. Studies suggest that NGF may help increase the survival of neurons that degenerate in AD. In this study NGF is delivered by surgical insertion into the region of the brain where cell death occurs.

**Requirements:**  
- 55-80 years old and in general good health  
- On stable AD medication for three months  
- Have a study partner for all visits  
- Fluent in English
ADRC Students, Staff, and Volunteers

Haileigh Smith and Trent Smith are twin siblings at High Tech High School who recently completed a one-month internship at the Shiley-Marcos ADRC as part of their junior year curriculum. The students worked with Dr. Jody Corey-Bloom on two research projects related to Huntington’s disease. They also attended our annual ADRC conference, visited the labs of our research scientists, and viewed a brain cutting done by Dr. Larry Hansen, an ADRC pathologist. The students sat in on nursing and neuropsychological evaluations and also attended the weekly early-stage support group to learn about the impact of dementia on both patients and families.

Trent and Haliegh had a grandfather with Alzheimer’s, so the issue is close to home for them. During their internship, they also did a search of websites and resources specifically directed to teens who have a parent or grandparent with dementia.

Their extensive resource list can be found on our website at: http://adrc.ucsd.edu/Resources_for_children_and_Teens.html

Jill Bansberg, BA is volunteering at the Shiley-Marcos ADRC at the suggestion of a professor and mentor at SDSU. Jill hopes to learn more about the symptoms of Alzheimer’s, how to provide better quality of life for the people affected, and how to participate in research that may contribute to their physical and emotional well being. “The loss of independence and memory is tragic for both the person affected as well as their families. I hope my experience here will help me to build skills to bring value and hope to the lives of people affected by AD in the future.”

Jill’s favorite part of her volunteer work is spending time with older adults. She provides much-appreciated assistance in our weekly Out and About program where she enjoys talking to the participants and hearing their stories, humor, and wisdom. She also attends our weekly early-stage support group and is collaborating with Dave Shay, a support group member with MCI, on a study investigating the role of humor in the support group. Jill is exploring graduate degree programs and wants a career in which she can make a positive contribution to those affected by Alzheimer’s. “I realized I had a calling to this particular field when I was working in an Alzheimer’s facility at an assisted living home about eight years ago. I began to appreciate and respect the power of the disease, but more so the strength of the people living with it.”

Joanne Gomez, BS joined our Shiley-Marcos ADRC team in March. She currently administers neuropsychological evaluations for the longitudinal studies and is also a part of our Shiley-Marcos ADRC Hispanic program. Joanne received her degree in Psychology from the University of California, San Diego. During her undergraduate studies, Joanne conducted research with Dr. Tamar Gollan on the effects of bilingualism in cognitive functioning by means of administering tests on Spanish-English bilingual undergraduate students. In addition, she collaborated with Dr. Keith Rayner on a recreation of the Paul A. Koler’s “Reading and Talking Bilingually” study by implementing Koler’s techniques and incorporating an Eyelink Eyetracker. Joanne is a fluent trilingual in English, Spanish, and French. She shares her spare time with her beloved canine companion, Cypress, and her loving family and friends. Her interests are hiking, music, cooking, and she recently picked up crocheting.
Elizabeth Choi and Gladys Ho are clinical psychologist trainees from the Chinese University of Hong Kong. Each summer since 2004, two students who have completed their Master’s degree training program in clinical psychology at the Chinese University of Hong Kong are selected to attend an eight-week practicum of observational and clinical research training at UCSD and the VA. Elizabeth and Gladys have previous experience in areas of child and adult psychiatric services, general medical hospitals and HIV clinics, and social welfare settings. Gladys previously worked as a research assistant in clinical trials for schizophrenia patients, and the psychological well-being of people with chronic grief. Elizabeth previously worked as a research assistant in the areas of neurocognitive outcomes of pediatric sleep apnea.

Both Gladys and Elizabeth hope to broaden their research knowledge on neurodegenerative diseases; learn neuropsychological assessment and interpretation; observe clinical services provided to Alzheimer’s patients and caregivers; and learn about cognitive rehabilitation services for patients with dementia. This training will help them to pursue further specialty training in neuropsychology and also improve clinical neuropsychological services for the aging population in Hong Kong.

Jin Deng recently graduated from La Costa Canyon High School and will begin his undergraduate education at the University of Chicago in the fall. He currently volunteers at the Shiley-Marcos ADRC to help enroll new participants into the various studies. Jin has also had the opportunity to observe neurologic and neuropsychological examinations, as well as various support groups. For him, the matter is personal. “My grandmother was actually diagnosed with AD in 2007, so I’m deeply appreciative of the opportunity to work in an environment where I can interact with others who are in her position and are coping with the same struggles she’s facing.”

Jin plans to earn a medical degree and eventually become a physician. “If there’s one thing I’m convinced of, it’s that any cure for Alzheimer’s disease can only be procured through more research, and I’d just like to assist with that process however I’m able to.” In his spare time, Jin enjoys adventuring, writing calligraphy, playing piano, golfing, and telling knock-knock jokes.

Sarah Espinoza, BA grew up in Mexico and returned to the US at the age of ten. She moved to San Diego in pursuit of her BA in Spanish Literature at the University of California San Diego. The past two years, she has focused her efforts at the Shiley-Marcos ADRC as a bilingual Psychometrist and now she takes on a position as a Hispanic Program Research Associate. As a Research Associate, she will continue her commitment to serving the Hispanic Program by closely collaborating with the nurses, acting as a clinical coordinator, and collaborating with the recruitment team. In addition, she will continue to carry out physician-referred community memory screenings at San Ysidro Health Center and in Chula Vista. She plans to use both her past and newly-gained knowledge to better serve the ADRC community. She has studied abroad in Latin America and through her travels and personal history she became interested in language acquisition and retention. Sarah has plans to pursue an MA in Speech Pathology. She loves to travel and has recently taken up archery and Son Jarocho, a traditional musical style of Veracruz, Mexico.
2012 SERIES

Memories at the Museums

SAN DIEGO MUSEUM OF ART
January 13, May 11, September 14

MINGEI INTERNATIONAL MUSEUM
February 10, June 8, October 12

TIMKEN MUSEUM OF ART
March 9, July 13, November 9

MUSEUM OF PHOTOGRAPHIC ARTS
April 13, August 10, December 14

Join us on the second Friday of each month from 2:00 - 3:00 at one of these exceptional San Diego museums for a unique docent-led discussion and tour. Museum docents engage people with mild-to-moderate Alzheimer’s or a related disorder and an accompanying family member or friend in discussions about the artwork to stimulate visual and verbal abilities and to spark memory. Memories at the Museums alternates between the four co-sponsoring museums that are all located in central Balboa Park. Museum admission and tours are free of charge to participants.