When individuals participate in our Shiley-Marcos Alzheimer’s Disease Research Center (ADRC), they are followed by our clinicians annually and receive complete physical, neurologic, and cognitive assessments in order to make as accurate a diagnosis as possible. Many families facing Alzheimer’s or a related dementia know, however, that the only way to make a definitive diagnosis of these conditions is by brain autopsy. Participants with dementia who enroll in our longitudinal study are asked to donate their brain at the time of death so that researchers can confirm diagnosis and better understand the impact of dementia on the brain. In the following interview, Dr. Masliah, an internationally renowned pathologist and valued member of our Shiley-Marcos ADRC team,

A Daughter’s View on Alzheimer’s, Autopsy, and Advocacy

BY FABIOLA MANRIQUEZ

When my father was diagnosed with Alzheimer’s in 2005, I was stunned and in disbelief. I had no idea what to anticipate for our future as a family dealing with an unexpected new challenge and the fears that accompanied it. As the daughter of an Alzheimer’s patient I felt lost, saddened, and frustrated beyond words. I asked myself, “Where would we be if help had not come our way?” Thankfully that question was short-lived due to the support system that has been provided by the Shiley-Marcos Alzheimer’s Disease Research Center (ADRC), especially, Frances Martinez – Goodrich, MSW (social worker). This program miraculously saved my family from drowning in a whirlwind of complexities from this debilitating illness. On behalf of my family, my step-mother, Martha Moreno, and my father, Jose Manriquez, I feel empowered and blessed to share our story, while acknowledging the importance of giving support to other caregivers, Alzheimer’s patients, and the community at large.
discusses his work with brain tissue and sheds light on the value of this significant donation. His contributions enable our scientists to have a more comprehensive understanding of the changes that occur as a result of the aging process and neurodegenerative conditions such as Alzheimer’s and related dementias.

How long have you been with the Shiley-Marcos Alzheimer’s Disease Research Center team?
I have been part of the UC San Diego Shiley-Marcos ADRC since 1988. I was recruited to UCSD by Drs. Robert Terry and Robert Katzman. For the past 22 years I have had the pleasure and honor to work with eminent neuroscientists at the ADRC including Drs. Saitoh, Thal, Koo, Galasko, Salmon and many others.

What do pathologists do?
There are two general branches of pathology: clinical pathology and anatomic pathology. Most pathologists are trained in both fields. Anatomic pathologists perform histopathological (tissue) analyses and provide diagnoses in biopsies and postmortem material, for example, diagnosis of cancer and other tumors. I am an anatomic pathologist. The clinical pathologists provide analyses and diagnoses in blood and other body fluids. All of laboratory medicine falls under the direction of a clinical pathologist. Examples of tasks performed by this type of pathologist include blood banking, tissue typing for transfusions or transplantation, and measuring enzymes in blood related to myocardial infarction (heart attacks).

Many persons think pathology is only dealing with the deceased, but it is so much more. The work of a pathologist has a direct impact on the diagnosis and treatment of many diseases. Moreover, the pathologist plays a key role in understanding the molecular and cellular mechanisms of disease, training of medical professionals, and quality assurance by checking for the accuracy and effectiveness of clinical diagnoses and treatment.

Why did you get interested in becoming a pathologist?
I have always been a very curious person and interested in diseases. I wanted to know how one cell kills another and what causes abnormal behavior in cells. I wanted to look at normal and abnormal cells under the microscope and see if they were physically different and then try to understand the root causes of what I saw. This led me to want to not only understand the causes of diseases but also develop new treatments for them.

In your tenure at the ADRC, what strides have been made? How does pathology contribute to the strides being made in AD research?
The only definitive way to diagnose Alzheimer’s and other related neurodegenerative conditions is by neuropathological analysis. Correlating the clinical and pathological findings is critical in trying to understand the pathogenesis of Alzheimer’s and other related neurodegenerative conditions.

In my laboratory at the ADRC, as we search for a cause of Alzheimer’s, we use mice that have been genetically engineered to mimic some of the pathological features of the disease (for example plaques and tangles) and test the effectiveness of treatments.

We have discovered two new molecules that might halt the progression of Alzheimer’s and Parkinson’s in mouse models and are planning to translate these findings to clinical trials.

For example for many years it was suspected that the amyloid plaques cause the dementia in Alzheimer’s disease. However, the neuropathological studies that we conducted discovered that it is actually the damage to the connections (synapses) among neurons that is most important, and that this is caused by small protein aggregates rather than by the actual plaques. Another interesting and important finding accomplished via these clinico-pathological correlations is the discovery that Alzheimer’s and Parkinson’s diseases overlap in a new con-
tion named Dementia with Lewy bodies (DLB). DLB has become a major focus of ADRC clinical research, as well as the molecular biological research done in my lab.

Why are autopsies important? In the clinical setting, the autopsies provide the definitive diagnosis and cause of death. They are also necessary to provide quality assurance for our clinical assessment process, our clinic-pathological correlations. Autopsy tissue and stained sections of tissue are also used for teaching medical students, residents and post residency fellows. Autopsy results provide education to the general public as to the causes of death and are often used for epidemiological studies. Of course this is in addition to providing closure to the family. A very important use of the tissue itself is to provide valuable material for research purposes representing a certain disease or as control tissue, without known disease.

In the last year, I have used tissue from ADRC autopsies for work on Dementia with Lewy bodies, comparing the severity of the neuropathology with age and also dementia severity. Another study involved the quantification of small aggregates of amyloid (mentioned above) and also synaptic proteins in different regions of brain tissue from both control and Alzheimer’s research subjects. We found that levels of a specific size of aggregates correlated with the severity of cognitive impairment and also with levels of specific kinds of synaptic proteins. These are just two of my recently published projects using tissue from ADRC participants.

In collaboration with others at the ADRC, we have identified the synapse as the point where the cascade leading to Alzheimer’s and other related neurodegenerative disease starts. Based on this work, we have developed experimental models in mice that have helped us to better understand how the synapse is damaged by small protein aggregates and how to use these mouse models to develop new treatments. We have currently discovered two new small molecules that might halt the progression of Alzheimer’s and Parkinson’s disease in mouse models and are planning to translate these findings to clinical trials.

What questions are you still trying to answer? The most critical point to resolve is the role of small protein aggregates in the mechanisms that damage neuronal connections and then use this to develop new treatments. We have identified new targets for intervention with this strategy and also new potentially therapeutic compounds. We want to understand if our compounds will halt the progression of Alzheimer’s and other related neurodegenerative diseases.

What do you do for fun? I enjoy spending time with my family; we enjoy visiting museums, attending concerts and going to the movies.

We hope that this interview has provided our readers with a more comprehensive understanding of the value that their brain donation has to the scientific advancement in the arena of Alzheimer’s disease and related dementias. Without the ability to perform an autopsy of the brain at the time of death, the value of the clinical data collected in the longitudinal study over such a prolonged period of time is limited. We sincerely appreciate our volunteers’ willingness to make this most precious donation.
I feel regardless of how, when, or where a person becomes an advocate for research, we all have a common denomination our passion for knowledge and its application. Ms. Martinez-Goodrich wanted to learn more about Alzheimer’s and joined the ADRC team in 1995, bringing with it, her passion to enlighten the Hispanic caregiving community and their families. Dr. Leon J. Thal, former Director of the ADRC, translated his passion and brilliance into the fields of Neurosciences and Alzheimer’s research. His amazing legacy and vast body of study helped transform his investigations into greater understanding of treatments for Alzheimer’s. Moreover, Dr. Doug Galasko, MD, the current co-director of the ADRC, continues to diligently strive to further advances in Alzheimer’s research. He is readily available to answer all of our questions at the annual ADRC Hispanic Caregivers Open House luncheon, while at the same time giving us updates on Alzheimer’s research. After learning so much about the difference these people make, I began to question what in turn I, as a Hispanic woman, can do to help advocate support for the ADRC. More specifically, what could be done for the Hispanic community and the Alzheimer’s research movement?

My first step towards activism began in February, 2007 with convincing my father to consent to donate his brain to the ADRC at the time of his death. Initially he resisted because of his religious beliefs, but after careful and delicate clarification, he consented. I further explained to him that we needed to increase the Hispanic participation within the research arena and that his contribution would have an everlasting impact towards finding a cure for Alzheimer’s. My involvement has taken on many forms in an effort to increase awareness. It began with a photo of my father, Martha, and I depicted on a flyer aimed at creating awareness of the ADRC’s outreach program to the public. Then in August 2007, I gave a presentation at the eighth annual Hispanic conference where I encouraged caregivers and their families to take care of autopsy consent forms and the advanced medical directives paperwork. My father, Martha, and I carry the autopsy consent card with us at all times. It has my fathers’ signature, a brief consensual acknowledgement of the autopsy, and two important phone numbers to call upon his transition.

It is best to embrace and prepare for this hardship rather than to ignore and delay the inevitable. I cannot emphasize enough, to every family, the importance of educating yourselves and learning how to prepare for a difficult and delicate medical challenge. I hope my words will support and guide every reader to overcome the uncertainties and embrace further education by becoming proactive and supporting this movement.
On March 9th, before a packed room in the Sacramento Capitol, the California Alzheimer’s Disease Advisory Committee and the Alzheimer’s Association presented the completed California State Plan for Alzheimer’s Disease (AD). The plan is a 10-year course of action with goals, recommendations, and strategies to prepare California for the impending catastrophic increase in Alzheimer’s and related dementias. The Alzheimer’s Association predicts a doubling by 2030 of Californians between the ages of 55 and 74 living with Alzheimer’s to 1.1 million. The first baby boomer turns 65 this year; it is estimated that one in eight will have AD after the age of 65; one in two will get AD by the age of 85. There are 10 million baby boomers that are predicted to be affected. This is not a trivial number.

State Senator Elaine Alquist (D-San Jose), author of SB 491 which authorized the development of a State Plan in 2008, stated before the room of advocates, legislators, and the press, “The economic and human costs of Alzheimer’s disease will be insurmountable if we don’t act now. Procrastination is simply not an option. Our public and private leaders must take advantage of the opportunity we have with this plan to make choices that will alleviate the worst impacts of a disease that’s already unimaginably tragic.”

The State Plan is intended to streamline government functions, reduce costs, and increase efficiency through the effective use of existing resources. The Plan includes a commitment to research and public awareness education campaigns. It also includes improving detection, diagnosis, treatment, and care for individuals and families impacted by this disease and better training of healthcare professionals, including physicians. The Plan also asks the State to “establish a comprehensive approach to support family caregivers.” Many services, some of which are funded by the State, provide overlapping functions and are poorly coordinated across multiple State agencies and departments. The Plan seeks to realign programs to “create a coordinated State infrastructure that enhances the delivery of care.”

Joshua Chodosh, MD, from UCLA and the VA Greater Los Angeles Healthcare System, co-chair of the State Plan Task Force said, “Unless we invest in home and community based care, including the family caregiver, we are simply shifting care to nursing homes, emergency rooms, and hospitals which places an even greater burden on the State.” The cost to care for a dementia patient in a nursing home is 2.5 times greater than care for a person without dementia.

Given the ongoing California budget crisis, the State Plan engaged a number of financing experts who shared their frustration with “siloed” government funding streams, discriminatory eligibility requirements, under-funding of community and home-based services and the need for more personal responsibility to share in the cost of long-term care. These experts identified the avoidance of long-term institutionalization as the highest priority followed by improving financial integration among services, and the provision of training and support for family caregivers. The State Plan development was made possible by private and philanthropic support and now to implement the Plan, we need to be creative and seek more private sector support. Government alone cannot address all the issues related to dementia.

Going forward, the Alzheimer’s Advisory Committee will request the cooperation and participation of the California Council of the Alzheimer’s Association and other interested partners in developing the first of five two-year action plans to provide the basis for implementing the goals, recommendations, and strategies laid out in the State Plan.

Mary Sundsmo, MBA is the Program Director of the Shiley-Marcos Alzheimer’s Disease Research Center, President of the California Council of the Alzheimer’s Association, and member of the State Plan Task Force.
Why are we also studying Parkinson’s Disease at the Alzheimer’s Disease Research Center?

BY DOUGLAS GALASKO, MD

Parkinson’s Disease (PD), first described by Dr. James Parkinson in 1817 as ‘The Shaking Palsy’, is best known as a movement disorder; tremor, slowing of movement, stiffening of the limbs and shuffling are typical. What does this have in common with the disease described almost a century later by Dr. Alois Alzheimer, which leads to forgetfulness and progressive cognitive decline?

For a start, both are neurodegenerative disorders – progressive conditions in which specific types or regions of nerve cells stop functioning properly, degenerate, and die. In PD and Alzheimer’s Disease (AD), vulnerable nerve cells are found in the brainstem and the cortex of the brain. Under the microscope, aggregates of misfolded proteins occur in PD and in AD. In PD, the culprit is a molecule called alpha-Synuclein (ASYN), which forms small structures called neurites in nerve cell processes, as well as larger structures called Lewy bodies within nerve cells. This is analogous to the buildup of tau protein in neurites and tangles in AD. However, ASYN may cause damage by mechanisms similar to those used by the other villain of AD – amyloid beta protein, or Aβ. Both ASYN and Aβ form small aggregates consisting of assemblies of a few molecules, known as oligomers. These cannot be seen under the microscope, and are identified using biochemical methods. Much evidence points to oligomers as toxins, which are capable of damaging nerve cells, either by causing membrane damage or impairment of synapses (the sites where nerve cells communicate with one another). Studying how abnormal forms of ASYN result in impairment of nerve cell function may lead to findings relevant to how Aβ causes damage in AD.

Risk factors for both Alzheimer’s and Parkinson’s include aging, as well as genetic vulnerability. Alzheimer’s Disease is more common overall, affecting almost 10% of people aged 65 or over, whereas PD affects about 1-2% of people aged 60 or over. Inherited forms of early onset Alzheimer’s are uncommon, but can be linked to onset of symptoms in people in their 30s and 40s. Genetic alterations (mutations) in three genes cause early onset AD. One of these mutations involves a duplication (extra copy) of the gene for APP, which gives rise to Aβ – this duplication results in overproduction of Aβ. Alterations in at least 8 different genes have been identified as potential causes of early onset inherited PD. One of these is the gene for ASYN, and interestingly enough, one of the gene mutations that can be found is a duplication or triplication (extra copies) of the ASYN gene, which in turn result in overproduction of ASYN. So, overproduction of the culprit proteins is a common theme in AD and PD genetics.

Some patients with dementia show changes of both AD and PD in their brains. The co-occurrence of Aβ, tau, and ASYN pathology has been studied since the late 1980s. It appears that AD and PD changes in the brain can interact, and their effects on the symptoms that patients show are predictable. In patients in whom cognitive decline is early and prominent, with underlying AD pathology, the additional presence of ASYN pathology often leads to slowing of movement and walking, and also to unusual symptoms such as fluctuation – marked variation of attention or cognition – and visual hallucinations. This picture is referred to as Dementia with Lewy Bodies, or DLB. A sizeable percentage of patients with PD develop cognitive decline after years of movement difficulty, and the brain often shows severe ASYN pathology accompanied by AD. This clinical picture is referred to as Parkinson’s Disease with Dementia (PDD). The boundaries between DLB and PDD are not clear, and it is helpful to consider them as part of a spectrum.
A major research goal at the ADRC is to understand the interactions between aging, AD, and PD pathology. We are using a number of research approaches to make progress. Drs. Joanne Hamilton and David Salmon are applying neuropsychological tests that evaluate how specific pathways in the brain link different types of information into a coherent whole, and how this changes in people with AD, PD, and DLB. Dr. James Brewer is studying highly detailed MRI scans of the brain to identify areas that may show atrophy (‘shrinkage’) in AD, PD, and DLB, and to measure how well different areas are interconnected. Dr. Douglas Galasko and other investigators are studying imaging methods such as PET to detect chemical changes in the brain, and also to measure Aβ, ASYN, and other chemicals in cerebrospinal fluid. Weaving these cognitive tests and biomarkers together will provide an extremely detailed picture of how the brain is changing in AD, PD, and the overlap conditions of DLB and PDD. These efforts will help us to improve early diagnosis and understand how these disorders progress.

Several laboratory scientists at UCSD are working on mechanisms of Aβ and ASYN toxicity, including Drs. Subhojit Roy and Eliezer Masliah. In 2010, Dr. Roy published an exciting paper with novel findings that suggest a new mechanism for how ASYN may lead to impaired function of synapses. Dr. Masliah’s research several years ago showed that increasing Aβ in a mouse model of ASYN pathology could worsen the extent of the ASYN changes. Recent work from Dr. Masliah’s laboratory has identified a number of promising pathways or targets that could allow new drugs to be developed to decrease ASYN pathology. Although it can take years to translate basic research leads into drugs, our researchers have many collaborations and connections in place to speed up the pace of discovery.

Kelly Landy, BA, received the Ruth L. Kirschstein National Research Service Award for Individual Predoctoral Fellows. Her award provides 3 years of funding through the National Institute on Aging. During this time, she will investigate visuospatial deficits in persons with Dementia with Lewy Bodies (DLB) and Alzheimer’s disease (AD). Using cognitive testing and a relatively new neuroimaging technique (diffusion tensor imaging), she hopes to gain a better understanding of changes in the brain associated with visuospatial problems in these two patient groups. DTI, or diffusion tensor imaging, is a specific type of MRI scan. These scans provide information about white matter tracts in the brain by using information about the direction of water diffusion in brain tissue. White matter tracts are responsible for connecting different parts of the brain, so DTI scans can give us useful information about intact or damaged connections among different brain regions. DLB patients commonly experience problems with visual perception. We think that DLB disrupts visual processing at a very basic level within the visual system. This study will explore the relationship between visual perception (measured with cognitive tests) with information about white matter tracts in the brain (measured with a DTI scan). The project may help explain and clarify the anatomical basis for visual perception problems in DLB and AD patients.

For more information, contact Kelly Landy at: 858-622-5839.
Clinical Trials

Nerve Growth Factor

PRINCIPAL INVESTIGATOR: Michael Rafii, MD, PhD
TIME INVOLVED: 24 Months

DESCRIPTION: Nerve Growth factor (NGF) research is a phase 2 double-blind, placebo controlled study. The purpose is to test the safety, tolerability, and effectiveness of an experimental 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. The ability of NGF to prevent brain cell loss in AD animal models has led to delivering NGF to humans. In this study NGF is delivered directly by surgical insertion into the area of the brain where cell death occurs. Gene therapy is experimental and has not yet been approved by the FDA.

REQUIREMENTS:
- 55-80 Years old
- On stable AD medication for 3 months
- Have a study partner for all visits
- Fluent in English
- In general good health

CONTACT: Christina Gigliotti, PhD
(858) 622-5800 | cgigliotti@ucsd.edu
Ask for the “Cere-100” Study

Neuroimaging Initiative Grand Opportunity

PRINCIPAL INVESTIGATOR: James Brewer, MD, PhD
TIME INVOLVED: 18 Months

DESCRIPTION: We are studying the earliest memory changes that occur with aging and are seeking people between ages 55 and 90 who have a concern about their memory. We will screen their memory using a memory test, and if it is mildly abnormal, we will examine brain structure and function using Magnetic Resonance Imaging (MRI) and Positron Emission Tomography (PET). We will also draw blood and cerebro-spinal fluid to determine the best approach for early diagnosis of neurodegenerative disease, such as Alzheimer’s disease.

REQUIREMENTS:
- Have a reliable study partner
- Memory complaint by patient and/or study partner
- MMSE score between 24 and 30 (inclusive).
- Able and willing to undergo lumbar puncture and MRI
- In good general health

CONTACT: Helen Vanderswag, RNC, BSN
858 622-5800 | hvanderswag@ucsd.edu
Ask for the “ADNI-GO” Study

Immune Globulin Intravenous (Human) IGIV

PRINCIPAL INVESTIGATOR: Michael Rafii, MD, PhD
TIME INVOLVED: Approximately 25 months

DESCRIPTION: This study aims to evaluate the novel use of an agent, Immune Globulin Intravenous (Human), 10% that is approved in the United States to treat various immunodeficiency and auto-immune disorders. IGIV is a biologic agent with anti-inflammatory and immunomodulating properties containing human immunoglobulin G antibodies derived from the blood plasma of healthy donors. Passive immunization could provide a safe and effective alternative to active vaccination for the treatment of AD patients, providing a strong rationale for studying passive immunization with IGIV.

REQUIREMENTS:
- 50-89 years old (inclusive)
- Diagnosis of probably AD
- MMSE score between 16 to 26 (inclusive)
- Have a study partner for all visits
- Able to read and write in English
- Stable health and medications

CONTACT: Elizabeth Ortega, NP
(858) 677-1567 | ejortega@ucsd.edu
Ask for the “IGIV/GAP” Study

Clinical Trials Registry

Are you interested in clinical trials but don’t find one that suits you? You can now join the Shiley-Marcos ADRC registry to be placed on a list for future studies. Participants can be normal controls, can have a mild memory problem, or can be diagnosed with early-to-moderate Alzheimer’s. Call the ADRC at (858) 622-5800.
Two recent research studies suggest that bilingualism or multilingualism may protect the brain from the symptoms of Alzheimer’s disease (Bialystok et al., 2007; Craik et al., 2011). These researchers reported that people who “spent the majority of life, at least from early adulthood regularly using at least two languages” first reported symptoms of Alzheimer’s disease about 5 years later than monolinguals (those who only speak one language). The bilinguals in these studies spoke a variety of languages (Yiddish, Polish, Italian, Hungarian, and French) and mostly originated from Eastern Europe.

Here at the ADRC we are beginning to investigate if similar effects can be found in our Spanish-English bilinguals. Instead of comparing bilinguals to monolinguals we are using a more specific measure of degree of bilingualism. The goal is to see if greater degrees of bilingualism result in greater benefits. Preliminary data suggest that the answer to this question is yes!

“EDUCATION AND BILINGUALISM MAY FUNCTION IN SIMILAR WAYS TO WARD OFF, OR MAKE THE BRAIN LESS VULNERABLE TO THE EFFECTS OF ALZHEIMER’S DISEASE.”

Each year during their annual evaluation with us, Hispanic participants at the ADRC are asked to name pictures in both Spanish and English. We classified those who can name similar numbers of pictures in Spanish and English as “more bilingual,” and those who name many more pictures in one language than in the other as “less bilingual.” Like the researchers in Canada we found an association between bilingualism and age of onset of Alzheimer’s. People who had similar naming scores in both languages tended to report symptoms of Alzheimer’s at a later age than those who were not as bilingual. However, we only found this benefit of bilingualism in participants with less than a high school education. For highly educated participants, degree of bilingualism didn’t seem to have any effect. These results suggest that education and bilingualism may function in similar ways to ward off, or make the brain less vulnerable to, the effects of Alzheimer’s disease.

These studies have generated considerable interest in the popular press but it is not yet clear how or why bilingualism delays the symptoms of AD in some populations. Currently we are developing a new picture naming test to improve on our ability to assess degree of bilingualism, and to expand our studies to include speakers of multiple languages. Spanish is the 2nd language most commonly spoken in the USA followed by Chinese. We are currently looking for Mandarin-English bilinguals over 65 years old who might be interested in participating in our studies of how bilingualism, aging, and Alzheimer’s disease may interact to affect cognitive processing.

For information on our bilingualism studies, please contact Tamar Gollan at 858-622-5898 or at ucsdlanguage@gmail.com
The ADRC was pleased to offer San Diego area seniors the opportunity to participate in one of three memory screening events held recently at our research center in La Jolla. Participants received a brief 30-minute assessment, in English or Spanish, of memory and other cognitive skills and were given immediate written feedback about their performance on these objective test measures. For each participant, screening results provided a ‘snapshot’ of current memory function, addressing the likelihood that cognition was ‘typical’ or ‘atypical’ for their age. All individuals were encouraged to share results with their personal physicians, who could provide medical follow-up if results suggested greater than expected decline in memory function.

WHO ATTENDED?

News stories appearing in San Diego’s major newspaper and heard on local public radio in November, 2010 brought public attention to the value of memory screening for seniors with concerns about decline in their memory function. As a host testing site for the Alzheimer Foundation of America’s ‘National Memory Screening Day’ on November 16, 2010, the ADRC received a swell of nearly 300 calls from individuals interested in making an appointment for assessment of their memory! The tremendous call volume required the ADRC to add two additional days of screening and the assistance of 15 extra volunteer staff members from affiliated research programs at UCSD in order to accommodate all those seeking evaluation.

“The ADRC received a swell of nearly 300 calls from individuals interested in making an appointment or assessment of their memory!”

Altogether, more than 200 individuals between 50-90 years of age were evaluated over the three full days dedicated to memory screening. Some who participated had real concerns about their own memory, some joined a family member in the testing experience to help ‘normalize’ the assessment process and minimize anticipated anxiety in their loved one, and still others expressed interest in a memory “check-up” because they had a family history of dementia or a personal relationship with someone with dementia and wanted to reduce worry about their own occasional memory lapses.
Screening results as a whole suggested that approximately 65% of participants had entirely age-consistent memory function, another 25% had questionable or minimal memory decline beyond what would be expected for their age, and results for about 10% suggested stronger evidence of true ‘deficiency’ in memory. All attendees received written feedback about their performance, with strong encouragement to discuss the results with their personal physician, the one most knowledgeable about their medical history and conditions that might have an impact on cognitive function and therefore the person most qualified to treat and manage these conditions.

**Why Participate in Memory Screening?**

Such a positive response to the November media stories and ‘Memory Screening Day’ participation confirms the personal relevance to seniors of monitoring the health of their thinking skills (especially memory), just as they attend to the more standard indicators of physical health such as blood pressure, cholesterol and diabetes. Increased interest in ‘memory health’ may at least in part be fueled by greater awareness and understanding of memory changes associated with normal aging and with illnesses like Alzheimer’s disease. An explosion of scientific research directed toward prevention and treatment of dementia has shed light on the importance of early identification of changes in thinking that may signal the beginning of progressive neurodegenerative disease. Though there is still no perfect intervention (i.e. ‘cure’) for Alzheimer’s disease and related dementias, the research has helped clarify methods of optimizing cognitive as well as physical health and of minimizing risk for dementia as one ages. We wish to commend the proactive approach of seniors who choose to address, rather than avoid concerns about memory function, and who wish to learn about and apply knowledge of medical and lifestyle contributions to optimize brain health.

**Exploring Options for Follow-Up**

In addition to undergoing cognitive assessment as part of the ADRC-hosted memory screening day events, participants had the opportunity after their assessment to enjoy some light refreshment, speak with ADRC staff, and gather informational literature on a range of topics related to memory research and community services providing assistance to those with dementia. Participants who expressed interest in becoming a part of some ongoing memory research through our center and its affiliated projects are receiving follow-up calls to explore available options, which must be reviewed on an individual basis. Qualified individuals may have the opportunity to enroll in the ADRC Longitudinal program, as well as projects examining brain imaging and cerebrospinal fluid biomarkers associated with Alzheimer’s disease, and/or participation in a clinical intervention/treatment trial.

Thanks to all who contributed to such a terrific community service experience! We look forward to hosting more such memory screening events again in the future.

**New Resource**

*2009 Progress Report on Alzheimer’s Disease: Translating New Knowledge*

Each year the National Institute on Aging releases a progress report on Alzheimer’s disease (AD). This 2009 issue, released in December of 2010, summarizes recent AD research conducted or supported by the National Institute on Aging and other components of the National Institutes of Health in lay terms for a general audience. The 64-page booklet is available in PDF format as a free download via the Alzheimer’s Disease Education and Referral (ADEAR) website of the National Institute on Aging (http://www.nia.nih.gov/Alzheimers).

The publication includes a brief overview of AD and the brain and research advances including:

1. Recognizing the Scope of AD
2. Deciphering AD Biology
3. Discovering New Genetic Mechanisms
4. Preventing AD and Promoting Healthy Brain Aging
5. Detecting Disease Earlier
6. Developing Novel Therapeutic Approaches
7. Testing Therapies for Prevention and Treatment
8. Supporting AD Caregivers

This comprehensive and accessible booklet is a useful resource for professionals in the field as well as persons with memory loss and their caregivers.
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 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 the charge to participants and are scheduled as follows:

SAN DIEGO MUSEUM OF ART  SEP 9
MINGEI INTERNATIONAL MUSEUM  JUN 10, OCT 14
TIMKEN MUSEUM OF ART  JUL 8, NOV 11
MUSEUM OF PHOTOGRAPHIC ARTS  AUG 12, DEC 9

Each monthly docent tour is limited to 8 pairs (16 participants total). Pre-registration is required. Please call Lisa Snyder at the Shiley-Marcos Alzheimer’s Disease Research Center at (858) 622-5800 to register for a tour.