When we get sick, we often wonder if stress had anything to do with it. In some cases, we are pretty sure it did. The realization that chronic stress can have serious health consequences can leave us puzzled as to what action to take to minimize the negative consequences that may occur. The research shows that there are individual differences in responses to stressful experiences, and that a response to a single stressful event can be very different from responses to stress that persist over time. The good news is, healthy responses to stress can be learned.

While most people are familiar with the contributions of stress to common medical problems such as heart disease, learning that chronic stress is a risk factor for cognitive problems and dementia may come as a surprise. My colleagues and I at the UCSD Shiley-Marcos Alzheimer's Disease Research Center (ADRC) have completed the first phase in a series of

(Continued on Page 2)
THE SPECTER OF CHRONIC STRESS: SHOULD WE BE CONCERNED?

(Co-Continued from Page 1)

studies that address the effects of chronic stress on cognitive functioning and risk for dementia. As reliable, scientific studies have established a link between stress and changes in regions of the brain associated with memory (e.g., the hippocampus), we have begun to recognize the potential importance of stress in the study of Alzheimer’s disease (AD), a disorder that typically begins in the hippocampus and surrounding regions of the brain. There is evidence to suggest that stress, particularly prolonged stress, increases susceptibility to damage in the hippocampus. Additional evidence supports a more direct link between stress and changes in the brain associated with AD.

Many of the initial investigations studied stress in animals and produced a wealth of ideas potentially applicable to humans. A number of these linked various types of stress to changes typically seen in the brains of individuals with AD (i.e., components of plaques and tangles). As evidence that chronic stress increases risk for cognitive decline began to accumulate, we sought to understand how this might happen as individuals grow older.

Our first project addressed the effects of chronic stress on memory at the time subjects entered the study (Peavy et al., 2007). We found that those who had not experienced highly stressful events over the previous year performed better on tests of memory than subjects exposed to highly stressful events (e.g., death of a family member, diagnosis of a potentially fatal illness). In addition, the data showed that stress level and possession of a specific genetic feature (Apolipoprotein E, e4 allele) previously identified as a risk factor for AD worked together to determine performance on memory tests. That is, for subjects divided into groups according to high or low stress level and presence or absence of the e4 allele, the group of individuals who possessed this genetic feature and were exposed to highly stressful events consistently showed the worst memory performance. These findings suggest that memory loss in older, independently-functioning adults is likely to result from the co-occurrence of prolonged exposure to stress and genetic make-up.

Our next project was designed to follow our research participants over the course of several years (Peavy et al., 2009) to determine how memory performance changed as a result of stressful experiences and level of the stress hormone cortisol. As expected, the presence of highly stressful life events was associated with faster decline on selected tests of memory in those subjects already experiencing some mild memory loss. However, for subjects with normal cognition at their initial visit, life stress ratings were not associated with accelerated cognitive decline. Because we expected that sustained higher levels of cortisol would be associated with cognitive dysfunction, our next finding came as a surprise. Participants with mild memory loss at their initial visit and higher averaged levels of cortisol declined at a slower rate than expected on tests of memory; this was not true for those who entered the study with normal memory. These findings suggest that the effects of cortisol on brain regions that regulate memory are different for subjects who are cognitively normal when compared to those who have mild cognitive impairment. The reason for this finding is unclear, but the higher levels of cortisol may have allowed subjects to compensate for their existing memory problems by improving their attention to the details of the memory tests. It is also possible that the participants were aware of deficits and increased their efforts to maintain their current functioning. Finally, it is possible that the extent of damage to specific brain regions (e.g., hippocampus) determines the degree to which high levels of cortisol can have toxic effects.

Our most recent findings address decline in functioning that signals a change of diagnosis (mild impairment non-demented to AD dementia) and support the results just described. We found that abnormalities in level of
cortisol, not the number of highly stressful events, were associated with worsening of cognitive functioning (particularly memory) when cognition was initially normal. However, when cognition was initially impaired, the number of highly stressful experiences, not cortisol levels, was associated with cognitive decline consistent with progression to a diagnosis of AD dementia. The consistency of these findings is encouraging given the ultimate goal of identifying ways to reduce risk and slow progression at different stages of susceptibility to debilitating changes and loss of independence.

The studies described here represent a beginning in our pursuit of information concerning the effects of chronic stress on cognitive functioning and the development of dementia in older adults. Interests for future studies at the ADRC include identification of additional stress-related neurochemical changes that affect cognitive functioning and determination of individual differences in personality characteristics and coping strategies in response to stressful experiences. Finally, we plan to use the results from these studies to identify specific behavioral interventions (e.g., changing habitual patterns of negative thinking) that are effective in reducing the harmful effects of chronic stress.

So, what about our original question that asked if we should be concerned about stress? The answer is yes, particularly during times of highly stressful experiences that persist over a significant period of time or for those individuals who respond to threat with significant distress and a sense of doom. Many of the risk factors for AD cannot be modified (e.g., age, genetic features). However, to minimize harmful effects of stress, responses can be altered using behavioral techniques (for example, progressive relaxation, shifts to positive thinking). Importantly, with some effort, modifying responses to stress can be learned at any age. As we address our research questions concerning these practical approaches, we will continue to communicate our findings with the hope of reducing vulnerability to Alzheimer’s disease. Your participation in our studies makes our research possible and is greatly appreciated.

altering its activity so that smaller amyloid beta (aβ) peptides are produced from the precursor protein app. Their finding was significant because longer (42 amino acid) aβ peptides are more likely to form toxic oligomers and lead to the formation of amyloid plaques (one of the hallmarks of Alzheimer’s disease). But, blocking all gamma-secretase activity will interfere with other transmembrane proteins cut by gamma-secretase, including notch. We know that blocking notch cleavage can cause potential severe adverse effects. Molecules with this type of activity, now called gamma-secretase modulators, have considerable theoretical advantages over gamma-secretase inhibitors.

As evidenced by the work awarded by MetLife, Dr. Koo’s lab focuses on understanding the pathophysiology of Alzheimer’s disease, with the hope of translating findings from basic cell and molecular biological studies to the clinical setting, to better understand the causes of the disease or have an impact on treatments. Koo has also been recognized for his studies on characterizing the pathways of production of the amyloid beta-protein from the amyloid precursor protein (APP). He has investigated the physiological function of the APP and how it might contribute to Alzheimer pathogenesis in ways unrelated to amyloid production. More recently, he has focused his attention on how synapses are damaged in Alzheimer’s disease.

The MetLife annual award for Alzheimer’s disease research has become a prestigious one in the field and a milestone in many researchers’ careers. The MetLife Foundation has granted major awards to scientists who have demonstrated significant contributions to the understanding of Alzheimer’s disease since 1986 with the goal of specifically recognizing the importance of basic research. This year, each winner received a $100,000 research grant and personal prize of $25,000 to further their work.

To learn more and to watch a video about Dr. Koo’s lab and work, please visit our website at http://adrc.ucsd.edu/thismonth.html
One of the most challenging parts of participating in the Alzheimer's Disease Research Center is completing difficult tests of memory and other thinking or "cognitive" abilities. We are often asked “Why do I have to take these tests?” and “What do you learn from them?” I would like to try to answer those questions now.

As you may know, the clinical symptoms of Alzheimer’s disease begin slowly and are often barely noticeable in the early stages of the disease. Also, the symptoms can vary from person to person. Although memory loss is usually the first symptom of the disease, changes in other cognitive abilities such as language, decision making, or the ability to visually recognize common objects or locations can occur early in the disease and in some cases may be more prominent than changes in memory.

Unfortunately, there are currently no simple biological tests that can detect Alzheimer's disease without looking for microscopic changes in brain tissue. Therefore, clinicians must diagnose the disease in most patients by carefully observing its clinical signs and symptoms and distinguishing them from changes that can occur as a normal part of aging. Because the initial changes of Alzheimer’s disease are very mild, detecting them can be difficult unless the patient’s abilities are challenged. Just as a treadmill test is often needed to challenge heart function in order to detect mild heart disease, the ADRC's difficult cognitive tests are needed to challenge a patient’s cognitive skills when early Alzheimer's disease is suspected. This is particularly true when a patient may have strong communication and social skills that can mask or “correct for” their cognitive changes so that the changes are not easily detected in normal conversation or even in an interview with a physician. By comparing a patient’s performance on very controlled and objective cognitive tests to the performances of groups of healthy people from their own age group, the clinician can decide with some certainty whether or not a “true” memory or other cognitive deficit exists.

Another important role for cognitive testing is to help the clinician decide whether the signs and symptoms shown by a patient are likely to be due to Alzheimer’s disease or to some other brain disease. Many diseases of the brain can cause problems with memory or other aspects of thinking, and sometimes these problems can be very similar to those that are caused by Alzheimer's disease. In addition, the earliest cognitive changes in Alzheimer's disease can vary from person to person making it even more difficult to decide if Alzheimer’s disease or some other disease is the culprit. Fortunately, research carried out by neuropsychologists over the past several decades has identified patterns of cognitive changes that are more likely to occur in Alzheimer's disease than in other brain diseases. By examining how a patient performs on tests of memory compared to tests of language, or on tests of decision making compared to tests of the ability to recognize common objects, the clinician can gain
confidence that the patient has early Alzheimer’s disease or has a different brain disease that can cause similar but subtly different cognitive problems. Unfortunately, detecting these distinct patterns of cognitive changes requires that each of the patient's cognitive abilities be thoroughly and rigorously tested and this increases the length and difficulty of the testing session.

Because Alzheimer's disease is a degenerative brain disease, memory and other cognitive abilities decline over time. However, this decline occurs at different rates in different people and can sometimes be slow and difficult to detect by the patient’s friends and family or even by their physician. In addition, different cognitive abilities may change at different rates. In one particular year, for example, a patient’s memory may not worsen while language or decision making abilities decline. The rate of change in cognitive abilities in a patient with Alzheimer’s disease, and the pattern of change over time, reflects how quickly and how widespread brain pathology is accumulating. Therefore, a third role for the ADRC’s challenging cognitive tests is to track the progression of the disease over time. By repeating testing each year, clinicians obtain an objective measurement of decline that can inform them of the disease's progress. This also allows the effects of potential treatments for Alzheimer’s disease to be evaluated. When the clinician knows the typical rate and pattern of cognitive decline that occurs in patients with the disease, any halt or slowing in this decline by a new medication can be detected.

Finally, the challenging cognitive testing carried out by the ADRC serves an important research function. The many benefits of testing that I have described would not be known except for years of research with dedicated patients and families who volunteer to complete testing year after year. This research has helped to identify the tests that are most effective in detecting the earliest cognitive changes that occur in Alzheimer’s disease, those that are most effective at tracking disease progression, and those that are most sensitive to medication effects. By comparing test performance with measures of brain degeneration on MRI scans, changes in biological factors in cerebrospinal fluid, genetic factors, and brain pathology seen at autopsy, investigators at the ADRC have identified how the biological changes of Alzheimer’s disease influence the memory and other cognitive changes that directly impact the patient’s life.

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**FREE MEMORY SCREENING FOR PERSONS WITH PARKINSON’S DISEASE**

**Do You or Someone You Know Have A Diagnosis of Parkinson’s Disease and Concerns About Memory?**

Schedule a **FREE**, 20 minute Memory Screening

**Friday, June 18, 9 a.m. to 4 p.m.**

- Instant results and feedback
- Speak with health professionals
- Additional information and resources available

**Shiley-Marcos Alzheimer’s Disease Research Center**  
8950 Villa La Jolla Drive, Suite C-129, La Jolla, CA 92037

Schedule your free 20-minute screening by calling **(858) 622-5800**. Participants must be 60 or older and have a diagnosis of Parkinson’s disease.

For more information, please visit [http://adrc.ucsd.edu](http://adrc.ucsd.edu) or call **(858) 622-5800**.

**Hosted by the UC San Diego Dementia with Lewy Bodies Program of the Shiley-Marcos Alzheimer’s Disease Research Center.**
### Clinical Trials

**Participating in Clinical Trials**

A clinical trial is a test or study of a new drug, device, or procedure. The following clinical trials are testing how effectively a medication works in relieving symptoms, diagnosing, or providing treatment for Alzheimer’s disease.

Although participation in a clinical trial does require some time commitment with visits to our Shiley-Marcos Alzheimer’s Research Center, in many cases, the visits are infrequent. Some people do not want to participate in a clinical trial if there is a chance of receiving a placebo (a look-alike pill with no medicinal ingredients).

It is well documented, however, that people who are unknowingly taking a placebo sometimes experience improvement of their symptoms or condition simply because they believe they are taking something that could be of benefit to them. Also, the ongoing support of the clinical trial coordinator can be a rewarding experience that increases feelings of well being for the participants.

Please contact us with any questions or concerns about our clinical trials. We greatly value your participation so that we can continue to make advances in the diagnosis, detection, and treatment of Alzheimer’s disease.

<table>
<thead>
<tr>
<th><strong>Nerve Growth Factor</strong></th>
<th><strong>BMS-708163</strong></th>
<th><strong>Passive Immunization-Amyloid Antibody Treatment for Alzheimer’s Disease</strong></th>
<th><strong>Alzheimer’s Disease Neuroimaging Initiative Grand Opportunity(ADNI-60)</strong></th>
<th><strong>Immune Globulin Intravenous (Human)</strong></th>
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<tbody>
<tr>
<td><strong>PRINCIPAL INVESTIGATOR</strong></td>
<td><strong>Michael Rafii, M.D., Ph.D</strong></td>
<td><strong>James Brewer, M.D., Ph.D</strong></td>
<td><strong>Michael Rafii, M.D., Ph.D</strong></td>
<td><strong>Michael Rafii, M.D., Ph.D</strong></td>
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<tr>
<td><strong>TIME INVOLVED</strong></td>
<td>24 Months</td>
<td>18 Months</td>
<td>18 months with at least 15 visits</td>
<td>Approximately 2.5 years</td>
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<tr>
<td><strong>DESCRIPTION</strong></td>
<td>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 a new 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 animal models of AD has led to delivering NGF to humans. In this study NGF is delivered directly by surgical insertion into the region of the brain where cell death occurs. Gene therapy is experimental and has not yet been approved by the FDA.</td>
<td>Identifying AD in the earliest phase of the disease process offers the opportunity to explore whether the use of potentially disease-modifying agents might alter the long-term course of the illness and prevent the neurodegenerative cascade associated with the disease. No drug therapy is currently indicated for prodromal AD. Studying the effect of BMS-708163, a potentially disease modifying agent, earlier in the disease process may have greater impact in the delay of progression of the illness.</td>
<td>A research study to learn if the investigational drug, bapineuzumab (AAB-001) is safe, well tolerated and effective for use in individuals with Alzheimer’s disease (AD). It is hoped that bapineuzumab will attach to amyloid in the brain and help remove it from the body. Participants will have a 60% chance of receiving the study drug vs a 40% chance of receiving a placebo (inactive drug). Throughout the study, participants will be monitored by a medical team of doctors and nurses.</td>
<td>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 autoimmune 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.</td>
</tr>
<tr>
<td><strong>REQUIREMENTS</strong></td>
<td>55-80 years old</td>
<td>Diagnosis of Mild Cognitive Impairment (not dementia)</td>
<td>Diagnosis of probable Alzheimer’s disease</td>
<td>Have a reliable study partner</td>
</tr>
<tr>
<td><strong>CONTACT</strong></td>
<td>Christina Gigliotti, Ph.D. at (858) 622-5800 and ask for the “Cere-110” study <a href="mailto:cgigliotti@ucsd.edu">cgigliotti@ucsd.edu</a></td>
<td>Helen Vanderswag, R.N.C., B.S.N. at (858) 622-5800 and ask for the “BMS” study <a href="mailto:hvanderswag@ucsd.edu">hvanderswag@ucsd.edu</a></td>
<td>Helen Vanderswag, R.N.C., B.S.N. at (858) 622-5800 and ask for the “ADNI-GO” study <a href="mailto:hvanderswag@ucsd.edu">hvanderswag@ucsd.edu</a></td>
<td>Elizabeth Ortega, N.P. at (858) 677-1567 and ask for the “IGIV/GAP” study <a href="mailto:ejortega@ucsd.edu">ejortega@ucsd.edu</a></td>
</tr>
</tbody>
</table>

**Clinical Trials Registry**

Are you interested in clinical trials but don’t find one that suits you? You can now join our Shiley-Marcos ADRC registry to be placed on a list for future studies.

**PARTICIPANTS CAN BE:**
- Normal Controls
- Have a mild memory problem
- Be diagnosed with early-to-moderate Alzheimer’s disease

Call the Shiley-Marcos ADRC at (858) 622-5800.

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**THERE ARE MANY NEW CLINICAL TRIALS AND RESEARCH PROTOCOLS ENROLLING AT THE SHILEY-MARCOS ADRC**

If you are interested in participating or would like more information, please contact the Study Coordinator listed with each trial.

- They can all be reached at the Shiley-Marcos ADRC - (858) 622-5800.
- There is no cost to participate in any of these research protocols.
- The Shiley-Marcos ADRC is under the direction of Douglas Galasko, M.D.

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- The participants.
- Increases feelings of well being for they believe they are taking some-toms or condition simply because of receiving a placebo (a look-alike pill with no medicinal ingredients).
- The visits are infrequent. Some people do not want to participate in a clinical trial if there is a chance of receiving a placebo (a look-alike pill with no medicinal ingredients).
- It is well documented, however, that people who are unknowingly taking a placebo sometimes experience improvement of their symp-toms or condition simply because they believe they are taking something that could be of benefit to them. Also, the ongoing support of the clinical trial coordinator can be a rewarding experience that increases feelings of well being for the participants.
- Please contact us with any questions or concerns about our clinical trials. We greatly value your participation so that we can continue to make advances in the diagnosis, detection, and treatment of Alzheimer’s disease.

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- Participating in Clinical Trials
- Nerve Growth Factor
- PRINCIPAL INVESTIGATOR: Michael Rafii, M.D., Ph.D
- 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 a new 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 animal models of AD has led to delivering NGF to humans. In this study NGF is delivered directly by surgical insertion into the region 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
- CONTACT: Christina Gigliotti, Ph.D. at (858) 622-5800 and ask for the “Cere-110” study cgigliotti@ucsd.edu

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- BMS-708163
- PRINCIPAL INVESTIGATOR: Michael Rafii, M.D., Ph.D
- TIME INVOLVED: 18 Months
- DESCRIPTION: Identifying AD in the earliest phase of the disease process offers the opportunity to explore whether the use of potentially disease-modifying agents might alter the long-term course of the illness and prevent the neurodegenerative cascade associated with the disease. No drug therapy is currently indicated for prodromal AD. Studying the effect of BMS-708163, a potentially disease modifying agent, earlier in the disease process may have greater impact in the delay of progression of the illness.
- REQUIREMENTS: 45-90 years old
- CONTACT: Elizabeth Ortega, N.P. at (858) 677-1567 and ask for the “BMS” study ejortega@ucsd.edu

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- Passive Immunization-Amyloid Antibody Treatment for Alzheimer’s Disease
- PRINCIPAL INVESTIGATOR: James Brewer, M.D., Ph.D
- TIME INVOLVED: 18 months with at least 15 visits
- DESCRIPTION: A research study to learn if the investigational drug, bapineuzumab (AAB-001) is safe, well tolerated and effective for use in individuals with Alzheimer’s disease (AD). It is hoped that bapineuzumab will attach to amyloid in the brain and help remove it from the body. Participants will have a 60% chance of receiving the study drug vs a 40% chance of receiving a placebo (inactive drug). Throughout the study, participants will be monitored by a medical team of doctors and nurses.
- REQUIREMENTS: 50 to 88 years of age
- CONTACT: Helen Vanderswag, R.N.C., B.S.N. at (858) 622-5800 and ask for the “ADNI-GO” study hvanderswag@ucsd.edu

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- Alzheimer’s Disease Neuroimaging Initiative Grand Opportunity(ADNI-60)
- PRINCIPAL INVESTIGATOR: James Brewer, M.D., Ph.D
- TIME INVOLVED: Approximately 2.5 years
- 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 standard memory test, and if it is mildly abnormal, we will examine brain structure and function using Magnetic Resonance Imaging and Positron Emission Tomography. 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: 50-89 years old, inclusive
- CONTACT: Elizabeth Ortega, N.P. at (858) 677-1567 and ask for the “IGIV/GAP” study ejortega@ucsd.edu

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- Immune Globulin Intravenous (Human)
- PRINCIPAL INVESTIGATOR: Michael Rafii, M.D., Ph.D
- TIME INVOLVED: Approximately 2.5 years
- 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 autoimmune 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
- CONTACT: Elizabeth Ortega, N.P. at (858) 677-1567 and ask for the “IGIV/GAP” study ejortega@ucsd.edu
Social Security Administration Adds Early-Onset Alzheimer’s to its Compassionate Allowances Initiative. On February 11, 2010, the Social Security Administration (SSA) announced that it will add early-onset Alzheimer’s disease (AD) to its Compassionate Allowances Initiative. This initiative identifies debilitating diseases and medical conditions that meet the disability standards for SSDI (Social Security Disability Income) or SSI (Supplemental Security Income). This initiative allows for faster processing and payment of benefits to individuals with AD.

An estimated 500,000 persons under the age of 65 are affected by Alzheimer’s disease. Many resources are designed for those over 65 years old, who can receive Medicare and Social Security benefits. The SSDI program is one of the few options available to these individuals.

In July 2009, a Compassionate Allowance Hearing on Early-Onset AD and related dementias was held in Chicago, including testimony from Harry Johns, President of the Alzheimer’s Association, researchers, caregivers, and individuals with early-onset AD. The Association also requested persons to submit written comments to SSA about their experiences applying for disability benefits. This proactive move by the Social Security Administration to ‘fast track’ certain conditions will reduce the backlog of disability claims and ensure that those with claims under this initiative won’t have to endure the financial and emotional toll of a long disability decision process.

Update from Capitol Hill. On March 9, 2010, more than 800 advocates traveled to Washington, DC and made more than 300 visits to members of Congress on Capitol Hill, sharing personal stories and statistics to continue the fight against Alzheimer’s disease. After a full day of training, we were prepared with three Alzheimer’s Association 2010 federal priorities. Once again we advocated for the Alzheimer’s Breakthrough Act (HR3286/HR1492) which would bring funding for Alzheimer’s disease research up to $2 billion at the National Institutes of Health. We also advocated for the Alzheimer’s Detection, Diagnosis, Care and Planning Act (ADD-CAP), providing Medicare reimbursement for bundled packages of services to increase diagnosis, and the National Alzheimer’s Project Act (S3036/HR4689), which would launch an inter-agency Advisory Council to create a coordinated National Alzheimer’s Disease Plan.

The Shiley-Marcos ADRC had three representatives on our San Diego advocacy team, two caregivers and me. The mood on the Hill seemed more positive than last year. We stressed the success of ‘investing in a disease’, such as was done for HIV, cancer and heart disease. These diseases now have declining mortality rates, while Alzheimer’s disease mortality grew from 2000-2006 by 46.1 percent. We didn’t ask our Congressmen to take funding away from them but rather to make Alzheimer’s disease a priority. We hope Congress listened.

YOU TOO CAN BE AN ADVOCATE!
Log on to: www.alz.org/join_the_cause_advocacy.asp and sign on to become an advocate or contact Pili Estall at the San Diego Chapter of the Alzheimer’s Association at Pilialoha.estall@sanalz.org or 858-492-4400.
Friday, February 26, 2010 was a beautiful day for the Hispanic Program Thank You Lunch. Sixty-two percent of those who called in to RSVP kept their commitment while another 15% who did not RSVP could not imagine missing the festivities and graced us with their presence. It was wonderful to see everyone.

Judith Rivera, MSN, FNP, our nurse practitioner, provided a brief overview of the program and various studies currently underway. She assured participants that regardless of recent budget cuts, they will still receive a call regarding their annual visit. She thanked them for being patient and working with us to schedule their appointments. She introduced Mary Margaret Pay, nurse practitioner and study coordinator of the Ceregene and the Home Based Assessment studies. Judith identified future ADRC clinical drug trials and applauded our Hispanics who accounted for 50% of those having participated in the Stem Cell biopsy study. Judith also welcomed Sarah Espinoza, psychometrist and our newest Hispanic team member who joined us following the departure of two of our very dear colleagues, Dr. Eileen da Pena and Rosa Montoya from our Hispanic program in 2009. We miss them.

Dr. David Salmon presented exciting news regarding a UCSD and SYHC (San Ysidro Health Center) coalition, THE MEMORY SCREENING CLINIC. Dr. Salmon touched on how brief mental status testing can be done in a primary care setting, but may under-detect true memory impairment in elderly patients with memory complaints. Based on the results of a previous Memory Screening Clinic model, the idea of UCSD and SYHC working together appears to be quite promising. He stated we will evaluate more people, and examine the effectiveness of our memory screening procedures for Spanish speaking elderly. If it is effective and useful, we will make it widely available to San Diego area physicians who treat Spanish speaking elderly patients. In closing, he expressed, on behalf of UCSD, our appreciation to SYHC physicians, patients and staff.

Tamar Gollan, Ph.D. provided some fascinating information regarding the bilingual brain and how it compares to that of a monolingual brain. According to Dr. Gollan, one of the first studies to compare bilingual and monolingual brains seemed to suggest that bilingualism leads people to develop denser brain tissue in some regions of the brain. She touched on how environmental demands can affect those who speak more than one language. Some studies seem to indicate that being bilingual may delay the onset of Alzheimer’s disease, although the effect may be stronger in people who speak more than two languages. She added; “It’s good to be bilingual!”

Dr. Aimee Pierce presented some of the “basics” regarding Alzheimer’s disease, the cause and current treatments. She touched on clinical trials for 2010 and emphasized how not all treatments are medications. Attendees were encouraged to consider getting three types of exercises on a daily basis:

1. Physical: 30 minutes, 5 days a week.
2. Mental: reading, puzzles, activities, and computer.
3. Social: friends, family, and support groups.

By Frances Martinez-Goodrich, MSW
Helpful Resources

**Can Alzheimer’s Disease Be Prevented?**

The recent National Institutes of Health (NIH) State-of-the-Science Conference on Preventing Alzheimer’s Disease and Cognitive Decline concluded that while there currently are no proven interventions to prevent Alzheimer’s disease, research points to several areas that merit further study. Is there anything people can do in the meantime to maintain a healthy brain and body? There are certain lifestyle choices and treatments that we know promote healthy aging and reduce risk of diseases like diabetes or cardiovascular disease. Studies suggest that some of the approaches for managing those conditions, like exercise or controlling high blood pressure, might also reduce the risk for Alzheimer’s disease and cognitive decline and are currently being tested in clinical trials. You can learn more in the National Institute on Aging’s (NIA) booklet, *Can Alzheimer’s Disease Be Prevented?*

To download or order free copies of *Can Alzheimer’s Disease Be Prevented?*, visit [www.nia.nih.gov/Alzheimers/Publications/ADPrevented](http://www.nia.nih.gov/Alzheimers/Publications/ADPrevented) or call NIA’s Alzheimer’s Disease Education and Referral Center toll-free at 1-800-438-4380.

**2008 PROGRESS REPORT ON ALZHEIMER’S DISEASE: Moving Discovery Forward**

Alzheimer’s disease research is moving forward in many scientific domains. Although progress can feel slow to those experiencing the impact of Alzheimer’s or a related disorder, each year progress is made that provides new knowledge to pave the way for advances in diagnosis, treatment, and perhaps one day, prevention of the disease. The National Institute on Aging (NIA) is the primary Federal agency supporting research in Alzheimer’s disease and age-related cognitive change. The NIA’s latest annual report, *2008 Progress Report on Alzheimer’s Disease: Moving Forward in Discovery*, summarizes current scientific directions and highlights findings from research funded by National Institutes of Health (NIH) through the year 2008. This report is now available online at [www.nia.nih.gov/Alzheimers/Publications/ADProgress2008](http://www.nia.nih.gov/Alzheimers/Publications/ADProgress2008). Due to budget cuts, single printed copies are not available.

**THE CALM BEFORE THE STORM**

Family Conversations about Disaster Planning, Caregiving, Alzheimer’s Disease, and Dementia

The recent earthquakes in Haiti and our own Northern Mexico and Imperial County areas remind everyone around the world that we are all vulnerable to the unpredictable and potentially tragic consequences of a natural disaster. Surviving and recovering from a disaster can create particular challenges for persons with Alzheimer’s or a related disorder and for their families. It is important that families consider strategies for managing potential natural disasters and have – to the extent possible – a plan of action in the event of an emergency.

The Hartford Financial Services Group and the Massachusetts Institute of Technology created this comprehensive 40-page booklet to help persons with dementia and their families better plan for natural disasters. You can download or order a printed copy at: [www.thehartford.com/calmbeforethestorm/index.html](http://www.thehartford.com/calmbeforethestorm/index.html).
HBO’s *The Alzheimer’s Project, Momentum in Science* - Companion Book Now Available

In May of 2009, HBO debuted their four-part documentary series, The Alzheimer’s Project. The four films each explored a different facet of Alzheimer’s disease. The first part, “The Memory Loss Tapes” highlighted the experience of memory loss from the perspective of the person with the disease. The second film, “Grandpa, Do You Know Who I Am?”, was designed to help children deal with the changes experienced by a relative with Alzheimer’s. The third two-part film, “Momentum in Science”, highlighted the current state of the art in scientific and medical advances in Alzheimer’s disease. The fourth and final film, “Caregivers”, provided insight into the experience of caring for individuals with AD at various stages in the process.

A companion book to the HBO Documentary Films series was recently released and is now available at no cost, while supplies last, at the Shiley-Marcos ADRC. The book specifically complements the material highlighted in the “Momentum in Science” film and explores the cutting-edge research on Alzheimer’s disease that is creating new hope for the future. It contains an in-depth look at the advances occurring in this area of research and helps the reader to better understand the cascade of events that occurs inside the Alzheimer’s brain, as well as the many approaches that are being undertaken by scientists to disrupt these processes and ultimately prevent the disease. The story that is told by the four-part book begins with a detailed overview of how the brain changes in Alzheimer’s disease, and methods in which it is currently diagnosed. From there, attention is given to advances in imaging techniques, as this area of research is holding great promise for early detection and diagnosis of the disease. The third part highlights the search for disease pathways to uncover the complex nature of the disease and the many ways in which scientists may be able to interrupt the cascade of events that ultimately leads to the manifestation of the disease. Finally, the book concludes with research that is aimed at changing the outlook, including chapters on studies that assess lifestyle factors, such as diet and exercise as well as the extensive array of studies focused on the development of new drug treatments for Alzheimer’s disease.

You may pick up your free copy of this hard-back book at the UCSD Shiley-Marcos ADRC while supplies last. Due to budget restraints, we cannot mail out books.

HOSPITALIZATION HAPPENS - A Guide to Hospital Visits for Individuals with Memory Loss

A trip to the hospital with a person who has memory loss or dementia can be stressful for all involved. This brochure can relieve some of that stress by helping you prepare for both unexpected and planned hospital visits.

Inside, you will find: steps you can take now to make hospital visits less traumatic; tips on making relatives or care partners more comfortable once you arrive at the hospital; and suggestions on how to work with hospital staff and doctors.

Keep this brochure in a convenient location and share this information with family and friends so you can begin preparing now for what could happen in the future.

You can download or order free copies of *Hospitalization Happens* by going to www.nia.nih.gov/Alzheimers/Publications/happens.htm
Memories at the Museums

This monthly program is in collaboration with San Diego Museum of Art, Mingei International Museum, Timken Museum of Art, and Museum of Photographic Arts. Museum docents guide visitors with mild-to-moderate Alzheimer’s and an accompanying family member or friend through the exhibits and provide a stimulating interactive experience. Memories at the Museums alternates between the four co-sponsoring museums and is entirely free of charge.

- **MINGEI INTERNATIONAL MUSEUM**
  - June 11

- **TIMKEN MUSEUM OF ART**
  - July 9

- **MUSEUM OF PHOTOGRAPHIC ARTS**
  - August 13

- **SAN DIEGO MUSEUM OF ART**
  - September 10

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.