



FREE PUBLIC LECTURE

Screening for Memory Impairment in the Elderly



Dr. David Salmon
Department of Neurosciences

Wednesday
July 20, 2005, 7:00 P.M.
Garren Auditorium
Basic Science Building
University of California, San Diego

NEXT MONTH

Immunity and Cancer

Dr. Thomas J. Kipps
Department of Medicine

Wednesday
August 10, 2005*, 7:00 P.M.
Garren Auditorium
Basic Science Building
University of California, San Diego

* Note date change.

No reservations required

Free parking will be provided for SIRA contributing members. Please call (858) 534-6299 TWO WEEKS BEFORE THE LECTURE to receive your free parking permit. All other lecture attendees must buy a permit at the parking kiosk on Gilman Drive or park at the metered sites.

Healthwise is available online at sira.ucsd.edu.

A Vaccine Approach to Treating Parkinson's Disease

Researchers at the University of California, San Diego (UCSD) School of Medicine working with scientists at Elan Pharmaceuticals, have reported promising results in mice of a vaccine approach to treating Parkinson's and similar diseases. These results appear in the June edition of the journal *Neuron*.

Eliezer Masliah, M.D., professor of neurosciences and pathology and SIRA faculty member at UCSD, and colleagues at UCSD and Elan Pharmaceuticals in San Francisco, vaccinated mice using a combination of the protein that abnormally accumulates in the brains of Parkinson's (called human alpha-synuclein) and an adjuvant. This approach resulted in the generation of anti-alpha synuclein antibodies in mice that are specially bred by Masliah's team to simulate

Parkinson's disease, resulting in reduced build-up of abnormal alpha-synuclein. The accumulation of abnormal alpha-synuclein is associated with degeneration of nerve cells and interference with normal inter-cellular communication, leading to Parkinson's disease and dementia.

The work marks the first time a vaccine for this family of diseases has been found effective in animal studies. Scientists at Elan Pharmaceuticals have been working for the past few years in a vaccine for Alzheimer's Disease.

The researchers focused on a spectrum of neurological disorders called Lewy body disease, which include Parkinson's and Alzheimer's. These disorders are marked by the presence of Lewy bodies—abnormal clumps of alpha-synuclein—in

continued on page 3

UCSD TV	PUBLIC LECTURE SERIES UCSD-TV SCHEDULE	Gene Therapy for Heart Failure Dr. Kirk Hammond	7/14 8:00 P.M.
			7/15 10:00 P.M.
			7/17 5:00 P.M.
			7/19 6:00 P.M.
		The Answer to Shingles Prevention Dr. Michael N. Oxman	7/28 9:00 P.M.
			7/29 11:00 P.M.
			7/31 5:00 P.M.
			8/2 7:00 P.M.

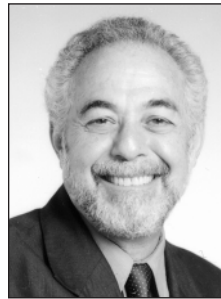
MORE OF SIRA ON UCSD-TV
Successful Aging Series

Sleepless in San Diego Dr. Daniel Kripke	7/21 8:00 P.M.
	7/22 10:00 P.M.
	7/24 5:00 P.M.
	7/26 6:00 P.M.

For clinical trials at UCSD: <http://health.ucsd.edu/ntrials/>

Something to Think About

J. Adam Milgram, M.A.



Dear Readers:

It is with some sadness, but also with great excitement that I have tendered my retirement notice to SIRA and UCSD. My last day of work will be Friday, August 5.

After seven years here as executive director, and editor of *Healthwise* and having reached the tender young age of 67+, I have decided to move on and follow what is true to my heart's desire.

For these many years, I have been the "face" of SIRA, and I have given my best to its positive functioning. I am more than satisfied with my efforts here, and feel that I have accomplished all that I can for SIRA and you, my readers. It has been my privilege to associate with so many wonderful people here—staff, faculty, and members of the Community Board of Advisors, and have thoroughly enjoyed our collaborative efforts. They have made my time here a period of personal growth and satisfaction, and have created a work environment that allowed for that. I am so grateful to all of them and you.

In my many articles, I have "preached the message" that life is a continuous opportunity from birth to death in which to develop and actualize one's inherent creative talents and abilities. Thus, I plan on retiring into a new life—rebirthing myself into the teacher, lecturer, writer, and inspirer of others in the psychospiritual dimensions of being, who are walking this most awesome path of life with me.

I want to thank you all from the bottom of my heart for your dedicated support of my efforts through the years. I intend to remain on the planet somewhere, and will be available if you need me. Coty Benrimoj, my most loyal assistant and friend here at SIRA, will always know how to contact me. Meanwhile, I can be reached at amilgram@ucsd.edu until August 5 and thereafter for a while at jamilgram@aol.com. My heartfelt love and blessings to all of you.

RECENT DONORS TO THE SIRA

We would like to express our deep appreciation for all those listed, as well as the anonymous donors, who chose to support the research, education, and patient care at the Sam and Rose Stein Institute for Research on Aging.

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Dear Readers :

Our next issue of Healthwise will be published in September 2005. Dr. Barry Lebowitz, deputy director of SIRA, will be the new editor. We hope to provide an even more informative and enjoyable newsletter.

Web Sites of Interest

www.healthcompass.org

Health Compass is a comprehensive site that helps older adults navigate the Internet for the best sources of health information. It features a glossary of common medical and research terms, checklists for patients to bring when they visit their doctors, and links to disease-specific medical organizations and support groups.

<http://www.hospitalcompare.hhs.gov>

The U.S. Department of Health and Human Services (DHHS) has launched this new Web site to provide consumers with information on how well hospitals care for adult patients. Hospital Compare uses voluntary, self-reported information that hospitals have submitted to the department regarding three major causes of morbidity and mortality—heart attack, heart failure, and pneumonia—to show how often the hospitals provide the rec-

ommended care for adults with one of those three conditions. Consumers can search for hospitals by state, county, city, zip code, or name.

<http://www.accentonseniors.com/index.htmh>

Accent on Seniors specializes in assisting families in their search for assisted living, nursing homes, board and care homes, independent/retirement living, and dementia care. They also help families in the Southern California area find senior products and senior services.

<http://aarp.co.mansellgroup.net/>

[UM/T.asp?A910.52852.2116.8.634456](http://aarp.co.mansellgroup.net/UM/T.asp?A910.52852.2116.8.634456)

AARP now offers a site where you can search or browse through an update directory of more than 900 carefully selected Web sites on aging.

A Vaccine Approach to Treating Parkinson's Disease

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the brain. Normally, alpha-synuclein proteins support communications between brain cells, or neurons. However, when abnormal proteins clump together in the neurons, a build-up of synuclein can cut off neuron activity, blocking normal signaling between brain cells and ultimately choking the cells to death.

"We found that the antibodies produced by the vaccinated mice recognized and reduced only the abnormal form of alpha-synuclein, since the protein's normal form is in a cellular compartment where antibodies can't reach it," said Masliah. "Abnormal alpha-synuclein finds its way to the cell membrane, where antibodies can recognize it."

Masliah stressed that the team's experimental active immunization, while effective in mice, may not be as useful in humans. "We would not want to actively immunize humans in this way by triggering antibody development, because one could create harmful inflammation," he cautioned. "However, it might be feasible to inject antibodies directly, as if the patient were creating his or her own."

While this research could take many years and holds no promise of prevention or cure, the researchers are hopeful that the mouse studies are a step in the right direction.

"This shows the first demonstration of a vaccine for this family of disease," Masliah said.

The research was supported by grants from Elan and the National Institute of Aging.

Bare Tree

Already I have shed the leaves of youth,
Stripped by the wind of time down to the truth
Of winter branches. Linear and alone
I stand, a lens for lives beyond my own,
A frame through which another's fire may glow,
A harp on which another's passion, blow.

The pattern of my boughs, an open chart
Spread on the sky, to others may impart
Its leafless mysteries that once I prized,
Before bare roots and branches equalized;
Tendrils that tap the rain or twigs the sun
Are all the same; shadow and substance one.
Now that my vulnerable leaves are cast aside,
There's nothing left to shield, nothing to hide.

Blow through me, Life, paired down at last to bone,
So fragile and so fearless have I grown.

—Anne Morrow Lindbergh

Interview with Dr. David Salmon

Department of Neurosciences

Editor: Welcome, Dr. Salmon. Could you tell us how you came to study memory impairment?

Dr. Salmon: I was trained in bio-psychology and was interested in the basic cognitive and neural processes involved in short-term memory in monkeys. I became interested in the biological basis of human memory and came to UCSD for a postdoctoral fellowship with the late Dr. Nelson Butters, who was one of the world's leading experts on the neuropsychology of memory. Dr. Butters studied a group of patients who had severe memory impairment due to a condition called alcoholic Korsakoff's syndrome. Due to long-term alcohol abuse and thiamine deficiency, these patients had damage in an area of the brain called the diencephalon that left them with very profound memory loss, or amnesia, affecting their ability to learn new information or to remember events from the past. We hoped to learn about the brain structures that were crucial for memory, and about the kind of memory processes that were affected.

At about the same time, interest was growing in another disorder that caused severe memory impairment in people, Alzheimer's Disease (AD). We wanted to see if their memory impairment was similar to other diseases that affected memory, and to understand the relationship between memory impairment and the brain structures that were damaged in AD. After I completed my fellowship, I joined the faculty here to continue that line of research and I am now the primary neuropsychologist for the UCSD Shiley-Marcos Alzheimer's Disease Research Center.

Editor: Where we are in our understanding of AD?

Dr. Salmon: There's a great deal of research on AD going on. As you know, AD is a disorder of progressive brain deterioration that leads to decline in memory and other mental abilities like language, problem solving, visual orientation, and attention. At a neuropathologic level, we know that there is a loss of nerve cells (or neurons) and synapses (the connections between nerve cells) in the brains of patients with AD, and that there are two types of abnormal lesions that occur, the neuritic plaque and the neurofibrillary tangle. We know that

these abnormalities occur in specific brain areas that are important for memory and other mental abilities, and that they disrupt normal cognitive functioning. We also are learning more about the basic biology of AD, including the components of the plaques and tangles, but we still don't know why plaques and tangles form or whether they cause cell death in the brain or are a result of cell death caused by some other factor.

We also now know that there can be a genetic component to AD. There are some families where AD is clearly inherited, but this is usually associated with early onset AD that occurs while a person is in their 30s or 40s. This clearly genetic form of AD is rare and occurs in only a very small percentage of people with the disease. In the usual case, AD doesn't develop until a person is in their 60s or 70s and there is not a clear pattern of inheritance. However, a genetic risk factor for the usual late-onset form of AD has been identified. Having a variant of the gene for something called apolipoprotein E increases the risk of developing AD as you get older. If you inherited one copy of this variant gene (the e4 variant) you have three times the risk of getting AD and eight times the risk if you inherited two copies.

Editor: Would you recommend people be tested for this variant gene?

Dr. Salmon: Although it is a risk factor, we generally don't think it has too much clinical value. If you think about it, even an eightfold risk of developing AD is not a huge increase and it certainly doesn't mean that everyone (or even most people) with the variant are going to develop AD. On the other hand, if you don't have the e4 variant it doesn't mean you're safe from AD.

Editor: What is your own research about?

Dr. Salmon: In general, my research is aimed at identifying the deficits in memory and other mental abilities that occur in patients with AD, and understanding how these changes are related to the pathology of the disease. My colleagues and I have shown that specific kinds of deficits in memory, language, problem solving, and spatial orientation occur early

in the course of AD, and how the presence of these deficits can be used to help clinically diagnose the disease. These particular deficits occur in AD because the pathological changes we talked about earlier, the cell and synapse loss and the plaques and tangles, occur in the brain areas important for these functions.

Editor: Problems with the frontal lobes?

Dr. Salmon: Yes, the frontal lobes, but also the temporal and parietal lobes. In fact, research shows that the earliest changes of AD typically occur in structures in the medial part of the temporal lobe known as the hippocampus and the entorhinal cortex. Interestingly, we know from Dr. Nelson Butters and Dr. Larry Squire, who is also here at UCSD, that these are brain structures that are critical for memory. So, it's not surprising that memory impairment is one of the earliest and most striking symptoms of AD.

Much of my current research is focused on reliably and effectively detecting AD as early as possible in the course of the disease. Early detection would allow potential treatments that might slow or halt the progression of AD to be applied as early as possible when they might be most effective. My colleagues and I at the UCSD Alzheimer's Disease Research Center have carried out long-term studies that included initially healthy, cognitively intact elderly individuals who went on to develop AD. Because we perform neuropsychological assessment of these individuals every year, we were able to go back and ask what their cognitive performance was like one, two, or three years before they received a diagnosis of AD. What we found was that their earliest cognitive change was almost always a decline in memory, and that this decline began several years before AD could be diagnosed by a clinician. In particular, we found that this early memory change was an inability to remember newly learned information over a short delay interval of 10 to 30 minutes. This so-called "rapid forgetting" may be the earliest reliable marker of the beginning stages of AD.

We have recently begun clinical research on the effectiveness and usefulness of memory screening in elderly patients who complain about memory problems to their primary care physicians. We have established a Memory Screening Clinic, funded by the State of California, as part of our research so that we

can perform a half-hour to forty-five minutes of cognitive testing and depression screening with this type of elderly patient and then report our findings back to the primary care physician for follow-up. Generally, we are able to tell the primary care physician whether or not our testing suggests that the patient has only memory impairment, memory impairment and other cognitive problems, symptoms of depression that could be contributing to their memory complaints, or no apparent deficits. If any of the first three conditions are suggested, we recommend that the primary care physician provides the patient with a more complete and thorough cognitive or depression work-up, and refers them to a neurologist or psychiatrist for evaluation. We found that 80 percent of the time when we said there might be a true problem, there was a note in the patient's chart that the primary care physician had discussed and explained the results to the patient, and/or had performed or referred them for additional work-up. We believe that a note of some action in 80 percent of cases is a very high percentage.

Editor: What about research in terms of cure?

Dr. Salmon: There are a number of scientists here at UCSD and around the world who are addressing the basic mechanisms of the disease. There are molecular and cellular studies, for example, that are trying to identify why plaques and tangles are formed in the brains of patients with AD, and what causes neurons to die. There are also studies trying to identify genetic factors that might be contributing to AD—attempting to identify what the gene actually does and perhaps somehow interrupt that process to stop disease progression or cure the disease. Although we still don't know the exact cause or causes of AD, we do know that the disease is very much a major public health issue. In the elderly, AD is possibly the third leading cause of death after cancer and heart disease. It also can greatly impact quality of later life. We all want to live as long and as successfully as possible while retaining our ability to remember, communicate effectively, and enjoy our environment.

Editor: Thank you.

Biomarkers in Aging, MCI, and Alzheimer's Disease

Dr. Galasko at the UCSD Alzheimer's Disease Research Center is conducting a study to measure the levels of a number of different proteins (called biomarkers) in cerebrospinal fluid (CSF) and in blood. The purpose of the study is to find out whether levels of these proteins are altered in people who have normal cognitive ability, mild memory problems, or early Alzheimer's Disease (AD). We aim to study these markers in detail to determine which combination of biomarkers is most helpful to indicate whether people with mild memory problems are at high risk of worsening over time, or progressing to AD. We will also test blood to find which form of a gene called Apolipoprotein E (APOE) an individual may have. Studies suggest that this gene may influence the risk of developing AD.

Recruitment is now underway to enroll subjects in one of the following categories:

- 1) healthy adults without memory problems, between ages 40–90
- 2) individuals with Mild Cognitive Impairment (MCI), between ages 60–90
- 3) individuals with early AD, between ages 60–90

The study will last five years and each year will involve a two-day (non-consecutive) visit.

During the first visit, participants will undergo brief physical and neurological examinations and will be asked a series of questions that are designed to evaluate memory, thinking capacity, and mood. They will also undergo laboratory (blood and urine) tests. The second visit will consist of further blood work and undergoing a spinal tap. Spinal taps, or lumbar punctures, are routine neurological outpatient tests in which a small amount of fluid is removed from the lower part of the back. The spinal taps are done to measure the biomarker proteins. Subjects will receive up to \$200 compensation per year of the study for undergoing the spinal taps. Subjects are not required to have a spinal tap each year if they choose not to do so. If you are interested in participating, call Helen Vanderswag, RN, at (858) 622-5805.

Noninvasive Method to Evaluate the Status of the Vascular System

Dr. A. Fronck has developed a noninvasive method to evaluate the status of the vascular system. The procedure is completely painless, takes about 40 minutes, and is approved by the UCSD Human Research Protection Program. Patients with past or present coronary disease (e.g., after bypass surgery or stents implants), patients with positive ekg results, patients with peripheral arterial disease, diabetes, hypercholesterolemia (treated or not treated), and hypertension, are encouraged to participate. Volunteers are invited to call (858) 534-4270 for additional information.

Brain Imaging Study

Sean Drummond, Ph.D., is conducting a brain imaging (MRI) study to determine how the brain reacts to lack of sleep. Participants must be 60 to 80 years of age and in good health. This study involves an overnight hospital stay at the VA Hospital in La Jolla and several brain imaging sessions. Participants will be given a physical exam, lab and ECG, a thorough sleep study, and are paid for their time and travel. If interested, please call Jen at (858) 642-1259.

Do You Have Age-Related Macular Degeneration and Feel Blue?

Do you have macular degeneration? Do you feel like your life is empty? Are you often bored? Do you feel hopeless? Dr. Stuart Brown and colleagues are currently screening volunteers for a study of escitalopram. This medication is not experimental. Escitalopram is an FDA approved antidepressant. The study is designed to see if the medication will help people with age-related macular degeneration adapt to living with this chronic condition. Subjects who qualify will receive study related care by board certified physicians. There is no cost to participate.

If you are interested in participating in this study or would like more information about the Age Related Macular Degeneration Outreach and Assist Programs, please call Linda Field at the Shiley Eye Center at (858) 822-1234 or (858) 822-2831.

UCSD Clinical Trials Center is Recruiting!

- Do you have COPD?
- Have you had a COPD exacerbation in the last 12 months . . . such as increased shortness of breath, increased sputum production, or change in the color of the sputum?
- Did you require either oral corticosteroids, antibiotics, and/or hospitalization to treat these symptoms?

UCSD Clinical Trials Center is conducting a national research study for COPD.

If you answered yes to the above questions, call Arlene at (619) 294-6239 or toll free at (888) UCSD-Air to learn about the study.

Dancer and White House Mini-Conference Delegate Catherine Baumgartner Reflects on the State of the Field

From *Creative Aging* newsletter, May 2005

Catherine Baumgartner is a member of Kairos Dance Theatre, a renowned inter-generational dance company. She is also a member of the Minnesota Creative Arts and Aging Network, based in Minneapolis.

Catherine is a delegate to the May 18–19 Creativity and Aging conference, an official mini-conference of the 2005 White House Conference on Aging co-sponsored by the National Endowment on the Arts, the American Association of Retired People, the International Music Products Association, and the National Center for Creative Aging.

In this interview, Catherine responds to questions about her career, about the state of the field of dance for older people, and the goals of the Creativity and Aging Conference.

Editor: What is your professional background?

Catherine: I have always been a dancer. I also worked with the Minnesota Dance Alliance for several years as a contract project coordinator. From 1999 to 2003, I worked with Marylee Hardenbergh, a site-specific choreographer. It was through Marylee that I met Maria Genne, the founder of Kairos Dance Theatre. I've developed quite a passionate interest in dance's broader relationship to community.

Editor: What interests you about dance and its relationship to community?

Catherine: I am interested in how dance allows communities to experience things through movement that can't be expressed in words. For example, people have said that seeing Kairos perform—seeing ages from 9 to 91 represented in our dances—is a reminder of what community could be like. The performance is more than the concept of community: dancing together is a way to experience community on an energetic and kinesthetic level.

Editor: How did you become involved in Kairos?

Catherine: In 2000, Maria was developing Kairos's first dance project for older adults in partnership with the Volunteers of America/Minnesota South West Senior Center. Maria asked me to be one of the facilitators.

Editor: How has working with Kairos impacted you as a dancer?

Catherine: Dancing with elders has added a good twenty-five years to my concept of what it means to be a vibrant older adult. That even if you move more slowly or with less range of motion, you can definitely still dance. I think of Kairos member Ocie May Young. She's 91, and the woman can boogie like you wouldn't believe!

Editor: As a delegate to the Creativity and Aging Conference, you have been researching the dance outlets available to older people in this country. What is your impression?

Catherine: I've been primarily looking into intergenerational dance companies that include older adults as performing members or that offer dance learning opportunities for older adults. My sense is that many senior centers across the country offer some kind of dance, for example line dancing or folk dancing. However, it seems that in most cases these programs use dance as a social outlet or a form of exercise. Dance programs for older people that explore dance as an art form exist, but there are fewer of them.

Perhaps this is because there's a widespread belief in our society that older people aren't capable of dancing. What's interesting to me is that I have the sense that almost the opposite is true in some cultural communities: elders are honored

as the "keepers" and teachers of dance, and they regularly dance in ceremonies and celebrations.

Editor: What other factors prevent dance outlets from being more widely available to older people?

Catherine: Training is key. Kairos often hears recreational therapists and activity directors say that they want to do dance work with older people but they don't feel qualified. They don't know dance as an art form. On the other side, professional dancers need to learn about working with older people. There are dance schools that offer adult dance classes, but these classes just aren't designed for people who might move a little more gently or a little slower. That isn't to say that there aren't people in their 70s and maybe even 80s taking flamenco or jazz dance—I'm sure they're out there too!

Another factor is funding. In order to attract funding, dance programs that target older people need to become more articulate to potential funders about the holistic benefits of dance. For example, there was a 2003 study in the *New England Journal of Medicine* that tracked older people in a range of leisure activities. Dance was the only physical leisure activity that correlated with a decreased likelihood of the onset of dementia (www.nejm.org). It's also key that we talk to funders about older people dancing not just from a health perspective, but from an artistic perspective.

Editor: What do you hope the Creativity and Aging conference will achieve?

Catherine: The field of arts and aging is a converging pathway between arts and health and social services, and it's up to us in the field to explain this convergence and its significance.



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