

# UCDAVIS

## ALZHEIMER'S DISEASE CENTER

The UC Davis Alzheimer's Disease Center conducts research and coordinates care for patients who have been diagnosed with Alzheimer's disease and other forms of dementia.

Summer Newsletter 2013

### The Alzheimer's Association's Fifth Annual African American Caregiving & Wellness Forum

By: Gwen Gates, BS



Welcome Message from Alzheimer's Association Chapter Board Member, Leslie Walker & Professor, Bruce Reed, PhD

For the past 5 years, the UC Davis Alzheimer's Disease Center has combined efforts with the Alzheimer's Association of Northern California to provide the African American Caregiver and Wellness Forum. With the support of the planning committee members from the Alameda County Area Agency on Aging and the office of Supervisor Keith Carson, we've brought in experts to provide vital resources and information on issues facing the African American Community. Such issues delve into the question of whether there is a connection between various health problems such as hypertension,

diabetes, strokes, heart disease, improper diet and decreased exercise, and their relationship to the onset of Alzheimer's disease. In addition, the forum provides the community with information on the latest research findings on Alzheimer's disease and related dementias.

The African American Caregiver and Wellness Forum has traditionally been conducted during the month of April or May of each year, and takes place at one of the senior centers in Oakland, CA, where many African American seniors frequently gather for various social and recreational activities.

On April, 2013, the African American Caregiver and Wellness Forum was held at the North Oakland Senior Center. Expert panelists answered questions and spoke on a variety of issues. Topics included: the connection between dementia and depression by Ladson Hinton, MD; a family approach to wellness by Pat Shanahan, MBA; financial planning by Tim Millar, MBA, CFP; patient rights & building partnerships by Monica Nowakowski-Carlson, LCSW; and stress reduction by Frank Staggers Jr., MD. Other presenters included Sherry

Tucker-Harris, MD; Bruce R. Reed, Ph.D.; and Leslie Walker.

Each year the forum has served as a means of creating a comfortable environment that allows caregivers and others to share their stories, ask important questions and get answers in a way that is clear, precise and thorough. This information is often

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Left to Right: Psychiatrist & Professor, Ladson Hinton, MD and a Wellness Forum attendee.



Attendees at the fifth annual African American Caregiving & Wellness Forum.



Internal Medicine Physician, Sherry Tucker-Harris, MD answering questions from the crowd.

For more information about future events, visit:

[www.alz.org](http://www.alz.org)

or call:

1-800-272-3900

## African American Caregiving & Wellness Forum

*Continued from front page*

from expert health care professionals from their own African American community. In addition, the forum has been instrumental in introducing the minority community to the field of Alzheimer's research and has helped in increasing minority recruitment and enrollment into various research studies that have in the past been underrepresented by the minority community. The forum has also been a means of giving back to the presently enrolled research participants of the UC Davis Alzheimer's Disease Center (ADC) in gratitude for the abundant amount of time and information that they have provided in the ADC's longitudinal research studies.

Throughout the past 5 years, attendance at the African American Caregiver and Wellness forum has ranged from 150 – 250 attendees. The success of this event is attributed to the personal outreach efforts of the planning committee, through presentations to churches, senior centers, senior housing facilities, mass mailings, e-mail blasts, media advertisement, flyer distribution, and word of mouth communication from the community itself. This outreach is extended throughout Alameda County, Contra Costa County and Solano County.

It is our hope that these forums will continue to be a vehicle for increasing minority participation and enrollment in research as well as reach more and more of the community to provide support and education to those with the disease, those caring for someone with the disease and those who are in need of information and resources about the disease. In addition, it is our hope that what is received from our forum attendees will promote positive behavioral changes and the eventual prevention of this devastating disease.

# Phase III Trial of Solamamezumab for Mild Alzheimer's Disease: Anticipation building for the first effective anti-amyloid therapy?

By: Dr. John Olichney & Dr. Berneet Kaur

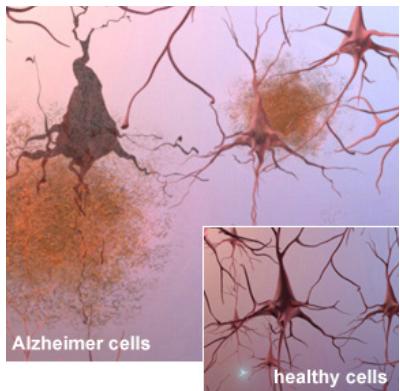


Image provided by Alzheimer's Association

Alzheimer's disease (AD) is the most common cause of dementia and the rates of AD in the American population are rising. At this time, the approved medications for Alzheimer's disease only treat the symptoms of the disease. We still have no treatments which slow down the underlying disease process, which involves microscopic plaques and tangles. The plaques are composed of a molecule called beta-amyloid, and research has focused on trying to decrease the amyloid plaques in the brain. The hope is that if we can somehow clear the amyloid from the brain, we actually change the disease itself (also known as disease-modifying therapy), or perhaps even cure the disease.

Research aims to treat the amyloid by using two major plans of attack. One is to use the body's immune system, the system that helps to fight off infections, to fight off the amyloid. One of the substances used is called intravenous immunoglobulin (IVIg). It is intravenous because it must be injected into the body from the veins. Another group of intravenous medications are called monoclonal antibodies. Examples of monoclonal antibodies include bapineuzumab or solanezumab. IVIg and bapineuzumab unfortunately failed to show significant results in Alzheimer's disease patients. Treatment with monoclonal antibodies is also associated with risks. The bapineuzumab study showed that a small number of patients had some brain swelling and even small amounts of bleeding (known as microhemorrhages).

Recently, a few bright rays of hope have emerged from studies of a new monoclonal antibody, called solanezumab. At the 2013 American Academy of Neurology meeting in San Diego, Dr. Anne Hake recently presented the results from the Expedition-1 and Expedition-2 trials of solanezumab, an Eli Lilly

study drug for mild to moderate AD. The initial results of this 76-week trial were negative. However, the researchers statistically "pooled" the patients with only mild AD from both Expedition trials. The "pooled" study showed significant improvements in memory and cognition. The results translated to approximately a 30% slowing of cognitive decline, and none of the subjects had the side effects of brain swelling or microhemorrhages. While this is not a cure, it may be an important disease-modifying therapy. If we could slow AD by 30% per year, after four years of therapy, this is equivalent to more than one year of delay in disease progression. This is greater than the combined effects of the available FDA-approved treatments for AD (cholinesterase inhibitors and memantine).

Monoclonal antibodies are expensive medications. As a society, we have to ponder questions such as: What are the economic and social benefits of slowing down AD progression, by let's say one year? Is an expensive treatment requiring regular intravenous infusions "worth it"?

The second method to attack beta-amyloid is by decreasing the amount of beta-amyloid the brain makes. Beta-amyloid is a normal protein in the brain, but as we get older or if we have a genetic risk, we may make more beta-amyloid than we need. The beta-amyloid then clumps together into the plaques. Research has shown that we can decrease the amount of beta-amyloid by decreasing the activity of the enzymes in the brain that make beta-amyloid. One of these types of medications is called a BACE-inhibitor. There are several BACE-inhibitors that are being developed for clinical trials. These medications are much easier to study, because they are taken by mouth. Future studies may even combine BACE-inhibitors with monoclonal antibodies to treat the amyloid plaques of AD.

*The UC Davis Alzheimer's Disease Center is always interested in new clinical trials and we welcome questions regarding approved clinical trials. We will also need volunteers when a new clinical trial starts.  
Please call Maria Levallois at (916) 734-5245 if you are potentially interested in participating.*

# The Parkinsonism and Dementia Connection

by: Dr. Berneet Kaur



*"If a patient with dementia is falling frequently, the patient's caregivers should ask their doctor about the possibility of parkinsonism."*

Many people have heard of Parkinson's disease, a condition which can cause tremor, rigidity and postural changes. In the later stages of Parkinson's disease, patients can develop a dementia, with cognitive changes much like Alzheimer's disease. However, other types of dementia also have mild "parkinsonism" – not Parkinson's disease

itself, but some of the subtle signs which are similar to Parkinson's disease. People often find this distinction confusing, which is understandable. However, it is important for doctors and patients to understand that people with both dementia and parkinsonism need special considerations.

Like Alzheimer's disease, there are no definitive tests for either Parkinson's disease or parkinsonism. These conditions are diagnosed at the bedside, by doctors experienced with these disorders. Doctors may observe that patients with parkinsonism move more slowly or have a tremor. Most importantly, these patients have instability when standing and walking, causing them to fall easily. Patients with dementia are already at risk of falling, resulting in broken bones and head injuries. Having parkinsonism in addition to dementia significantly increases the risk of falls. If a patient with dementia is falling frequently, the patient's caregivers should ask their doctor about the possibility of parkinsonism.

Alzheimer's disease is the most common cause of dementia and in the late stages of Alzheimer's disease, patients may have some mild parkinsonism. However, if parkinsonism occurs at the early or moderate stage of dementia, then the patient may not have Alzheimer's disease. One consideration is Lewy body dementia, a

dementia associated with parkinsonism and hallucinations in the early stages of the disease.

Another possibility is vascular disease of the brain, cerebrovascular disease. These patients may have had small, silent "strokes", called infarcts. Patients with cerebrovascular disease may also have chronic problems with blood flow through microscopic blood vessels of the brain, referred to as ischemia. Both silent infarcts and ischemia can result in dementia and parkinsonism.

## *The Best Treatment for Dementia and Parkinsonism is Prevention:*

- *Avoiding Strokes and Heart Disease*
- *Treating High Blood Pressure, Diabetes and High Cholesterol*
- *Getting Regular Exercise*
- *Maintaining Good Nutrition*

Certain medications can actually cause parkinsonism or make parkinsonism worse: medications for nausea like promethazine, or medications for psychosis, like haloperidol or risperidone. These medications should be avoided. Unfortunately, medications for Parkinson's disease, such as pramipexole, ropinirole or carbidopa/levodopa, may make symptoms of dementia and behavioral problems worse, without making the parkinsonism better. Physical therapy and the use of assistive devices, such as a walker, can be important for avoiding falls and improving mobility. Medications which cause dizziness as a side effect can also increase the risk of falls and should be avoided. Ultimately, the risks from parkinsonism in dementia cannot be eliminated entirely. However, awareness of the potential risks from unrecognized parkinsonism is key to avoiding and reducing its complications.



# The Effect of Diabetes on the Brain

**UC Davis researchers find that over-secretion of a protein secreted with insulin may play a role in Alzheimer's disease**

*"We've known for a long time that diabetes hurts the brain, and there has been a lot of speculation about why that occurs, but there has been no conclusive evidence until now."*

- Dr. Charles DeCarli

Neurology Professor, Director of the UC Davis Alzheimer's Disease Center



*"If we're smart about the treatment of pre-diabetes, a condition that promotes increased amylin secretion, we might be able to reduce the risk of complications, including Alzheimer's and dementia."*

- Dr. Florin Despa

Pharmacology Assistant Professor-In-Residence

A protein secreted with insulin travels through the bloodstream and accumulates in the brains of individuals with type 2 diabetes and dementia, in the same manner as the amyloid beta (A $\beta$ ) plaques that are associated with Alzheimer's disease, a study by researchers with the UC Davis Alzheimer's Disease Center has found. The study is the first to identify deposits of the protein, amylin, in the brains of people with Alzheimer's disease, as well as combined deposits of amylin and A $\beta$  plaques. These findings suggest that amylin is a second amyloid as well as a new biomarker for age-related dementia and Alzheimer's.

"We've known for a long time that diabetes hurts the brain, and there has been a lot of speculation about why that occurs, but there has been no conclusive evidence until now," said UC Davis Alzheimer's Disease Center Director Charles DeCarli. "This research is the first to provide clear evidence that amylin gets into the brain itself and that it forms plaques that are just like the amyloid beta that has been thought to be the cause of Alzheimer's disease," DeCarli said. "In fact, the amylin looks like the amyloid beta protein, and they both interact. That's why we're calling it the second amyloid of Alzheimer's disease." The journal article "Amylin deposition in the brain: A second amyloid in Alzheimer's disease?" published online in the Annals of Neurology.

Type 2 diabetes is a chronic metabolic disorder

that increases the risk for cerebrovascular disease and dementia, a risk that develops years before the onset of clinically apparent diabetes. Its incidence is far greater among people who are obese and insulin resistant.

Amylin, or islet amyloid polypeptide, is a hormone produced by the pancreas that circulates in the bloodstream with insulin and plays a critical role in glycemic regulation by slowing gastric emptying, promoting satiety and preventing post-prandial spikes in blood glucose levels. Its deposition in the pancreas is a hallmark of type 2 diabetes.

When over-secreted, some proteins have a higher propensity to stick to one another, forming small aggregates, called oligomers, fibrils and amyloids. These types of proteins are called amyloidogenic and include amylin and A $\beta$ . There are about 28 amyloidogenic proteins, each of which is associated with diseases.

The study was conducted by examining brain tissue from individuals from three groups: those who had both diabetes and dementia from cerebrovascular or Alzheimer's disease; those with Alzheimer's disease without diabetes; and age-matched healthy individuals who served as controls.

The research found numerous amylin deposits in the gray matter of the diabetic patients with dementia, as well as in the walls of the blood vessels in their brains, suggesting amylin influx from blood

*Diabetes and Alzheimer's Disease Continued on p. 6*



circulation. Surprisingly, the researchers also found amylin in the brain tissue of individuals with Alzheimer's who had not been diagnosed with diabetes; they postulate that these individuals may have had undiagnosed insulin resistance. They did not find amylin deposits in the brains of the healthy control subjects.

"We found that the amylin deposits in the brains of people with dementia are both independent of and co-located with the A $\beta$ , which is the suspected cause of Alzheimer's disease," said Florin Despa, assistant professor-in-residence in the UC Davis Department of Pharmacology. "It is both in the walls of the blood vessels of the brain and also in areas remote from the blood vessels. It is accumulating in the brain and we found signs that amylin is killing neurons similar to A $\beta$ ," he continued. "And that might be the answer to the question of 'What makes obese and type 2 diabetes patients more prone to developing dementia?'"

The researchers undertook the investigation after Despa and his colleagues found that amylin accumulates in the blood vessels and muscle of the heart. From this evidence, he hypothesized that the same thing might be happening in the brain. To test the hypothesis he received a pilot research grant through the Alzheimer's Disease Center. The research was conducted using tissue from the brains of individuals over 65 donated to the UC Davis Alzheimer's Disease Center: 15 patients with Alzheimer's disease and type 2 diabetes; 14 Alzheimer's disease patients without diabetes; and 13 healthy controls. A series of tests, including Western blot, immunohistochemistry and ELISA (enzyme-linked immunosorbent assay) were used to test amylin

accumulation in specimens from the temporal cortex.

In contrast with the healthy brains, the brain tissue infiltrated with amylin showed increased interstitial spaces, cavities within the tissue, sponginess, and blood vessels bent around amylin accumulation sites.

Despa said that the finding may offer a therapeutic target for drug development, either by increasing the rate of amylin elimination through the kidneys, or by decreasing its rate of oligomerization and deposition in diabetic patients. "If we're smart about the treatment of pre-diabetes, a condition that promotes increased amylin secretion, we might be able to reduce the risk of complications, including Alzheimer's and dementia," Despa said.

**Additional study authors are Kaleena Jackson, Gustavo A. Barisone, Elva Diaz and Lee-Way Jin, all of UC Davis.** The study was funded by National Science Foundation grant CBET 1133339 (F.D.); American Diabetes Association grant 1-13-IN-70 (F.D.); the University of California, Davis Alzheimer's Disease Pilot Project Program (F.D.); National Institute on Aging award P30AG010129 (C.D.); and a Vision Grant from the University of California, Davis Health System (F.D.).

The UC Davis Alzheimer's Disease Center is one of only 29 research centers designated by the National Institutes of Health's National Institute on Aging. The center's goal is to translate research advances into improved diagnosis and treatment for patients while focusing on the long-term goal of finding a way to prevent or cure Alzheimer's disease. Also funded by the state of California, the center allows researchers to study the effects of the disease on a uniquely diverse population.



## La Mini Escuela de Medicina en Español Regresa el Sábado 7 de Septiembre!

La sexta Mini Escuela de Medicina en Español anual - La primera de su estilo en la nación – Tomará lugar el sábado 7 de septiembre. En el primer piso del MIND Institute del Centro Médico de UC Davis (2825 50th St. Sacramento, CA). El evento será otra vez en español y presentará a profesionales de salud que darán conferencias sobre una variedad de temas relacionados a la salud del cerebro y el envejecimiento. Este evento es organizado por el UC Davis, Alzheimer's Disease Center, en conjunto con el Latino Aging Research and Resource Center y el Center for Reducing Health Disparities. Este evento está abierto al público, pero el espacio es limitado.

Llame a Rebekha Alfaro al (916) 734-5243 del Centro de la Enfermedad de Alzheimer para reservar su lugar.

# Investigadores de UC Davis encuentran que un segundo amiloide puede tener parte en la enfermedad de Alzheimer

*“Desde hace bastante tiempo, se sabe que la diabetes produce lesión cerebral, y hubo mucha especulación acerca de por qué ocurre esto, pero hasta el momento no existía ninguna prueba concluyente.”*

- Dr. Charles DeCarli

Director del Centro de la Enfermedad de Alzheimer de UC Davis



*“Si usamos un abordaje inteligente para tratar la prediabetes, una afección que promueve la secreción de amilina, podríamos estar en condiciones de reducir el riesgo de complicaciones, incluida la enfermedad de Alzheimer y otras demencias.”*

- Dr. Florin Despa

Profesor adjunto residente del Departamento de Farmacología de UC Davis

Un estudio realizado por los investigadores del Alzheimer's Disease Center (Centro de la Enfermedad de Alzheimer) de UC Davis halló que la proteína secretada con insulina viaja por el torrente sanguíneo y se acumula en el cerebro de las personas con diabetes tipo 2 y demencia, igual que las placas de proteína beta-amiloide ( $A\beta$ ) asociadas a la enfermedad de Alzheimer. Este estudio es el primero en identificar depósitos de esta proteína, conocida como amilina, en el cerebro de las personas que padecen la enfermedad de Alzheimer, como así también depósitos combinados de amilina y placas  $A\beta$ , lo cual sugiere que la amilina es un segundo amiloide, además de ser un nuevo biomarcador del mal de Alzheimer y otras demencias seniles.

“Desde hace bastante tiempo, se sabe que la diabetes produce lesión cerebral, y hubo mucha especulación acerca de por qué ocurre esto, pero hasta el momento no existía ninguna prueba concluyente”, dijo Charles DeCarli, director del Centro de la Enfermedad de Alzheimer de UC Davis. “Esta investigación es la primera en ofrecer una evidencia clara de que la amilina penetra en el cerebro y forma placas igual que la  $\beta$ -amiloide que se cree es la responsable de la enfermedad de Alzheimer”, agregó DeCarli. “De hecho, la amilina se asemeja a la proteína  $\beta$ -amiloide y ambas

interactúan. Es por eso que la denominamos el segundo amiloide de la enfermedad de Alzheimer”.

“Amylin deposition in the brain: A second amyloid in Alzheimer's disease?” se encuentra publicado hoy en Internet en Annals of Neurology.

La diabetes tipo 2 es un trastorno metabólico crónico que aumenta el riesgo de enfermedades cerebrovasculares y demencia, un riesgo que está presente años antes de la primera manifestación clínica de la diabetes. Su incidencia es mucho mayor entre las personas obesas y resistentes a la insulina.

La amilina, o polipéptido amiloide de los islotes, es una hormona secretada por el páncreas que circula por el torrente sanguíneo junto con la insulina y juega un papel fundamental en la regulación de la glucemia, ya que desacelera el vaciado gástrico, promueve la saciedad y previene los picos posprandiales en los niveles de glucosa. Su depósito en el páncreas es una señal clara de diabetes tipo 2.

Frente a un exceso de secreción, algunas proteínas son más propensas a adherirse a otras, y forman pequeños agregados, denominados oligómeros, fibrilares y amiloïdes. Estos tipos de proteínas también se denominan proteínas amiloidogénicas e incluyen a la amilina y a la  $A\beta$ . Existen cerca de 28 proteínas amiloidogénicas, cada una de ellas asociadas a

*Segundo Amiloide continúa pg. 8*

enfermedades.

El estudio se realizó mediante el examen del tejido cerebral de personas comprendidas en tres grupos distintos: sujetos con diabetes y demencia por enfermedades cerebrovasculares o mal de Alzheimer; sujetos con mal de Alzheimer sin diabetes; y personas de la misma edad que no presentaban patologías y que sirvieron como grupo de control.

La investigación halló numerosos depósitos de amilina en la materia gris de las pacientes diabéticas con demencia, así como también en las paredes de los vasos sanguíneos del cerebro, lo cual sugiere la entrada de amilina por la circulación sanguínea. Sorprendentemente, los investigadores también hallaron amilina en el tejido cerebral de las personas con la enfermedad de Alzheimer a quienes no se les había diagnosticado diabetes; postulan que es posible que estos sujetos hubieran tenido una resistencia a la insulina no diagnosticada. No se halló depósito de amilina en los cerebros de los sujetos sanos del grupo de control.

“Hallamos que los depósitos de amilina en el cerebro de las personas con demencia son independientes de y comparten la misma ubicación con la A $\beta$ , que se sospecha es la responsable de la enfermedad de Alzheimer”, dijo Florin Despa, profesor adjunto residente del Departamento de Farmacología de UC Davis. “Se encuentra en las paredes de los vasos sanguíneos del cerebro y también en áreas distantes de los vasos sanguíneos”. “Se acumula en el cerebro y hallamos signos de que la amilina mata a las neuronas en forma similar a la A $\beta$ ,” agregó. “Y ésta podría ser la respuesta a la pregunta: ¿por qué los obesos y los pacientes con diabetes tipo 2 son más propensos a padecer demencia?”.

Los investigadores siguieron la investigación de Despa y sus colegas descubrieron que la amilina se acumula en los vasos sanguíneos y en el músculo del corazón. A partir de esta evidencia, planteó la hipótesis de que lo mismo debería estar ocurriendo en el cerebro. Para confirmar la hipótesis, el Centro de la Enfermedad de Alzheimer le otorgó una subvención para investigación piloto.

La investigación se llevó a cabo utilizando el tejido cerebral de personas mayores de 65 años donados al Centro de la Enfermedad de Alzheimer de UC Davis: 15 pacientes con la enfermedad de Alzheimer y diabetes tipo 2; 14 pacientes con la enfermedad de Alzheimer sin diabetes; y 13 controles sin patologías.

Se utilizaron una serie de pruebas, entre ellas, el análisis de Western blot, la inmunohistoquímica y la prueba ELISA (prueba de inmunoabsorción enzimática) para analizar la acumulación de amilina en muestras de la corteza temporal.

A diferencia de los cerebros sanos, en el tejido cerebral infiltrado con amilina se detectó más espacios intersticiales, cavidades dentro del tejido, esponjosidad y vasos sanguíneos curvados alrededor de los lugares con acumulación de amilina.

Despa manifestó que el hallazgo puede ofrecer una meta terapéutica para el desarrollo farmacológico, ya sea mediante el aumento del índice de eliminación de amilina a través de los riñones, o mediante la disminución del índice de oligomerización y depósitos en pacientes diabéticos. “Si usamos un abordaje inteligente para tratar la prediabetes, una afección que promueve la secreción de amilina, podríamos estar en condiciones de reducir el riesgo de complicaciones, incluida la enfermedad de Alzheimer y otras demencias”, dijo Despa.

**Otros autores del estudio incluyen a Kaleena Jackson, Gustavo A. Barisone, Elva Diaz y Lee-Way Jin, todos ellos de UC Davis.** El estudio fue financiado por la Fundación Nacional de Ciencias, subvención CBET 1133339 (F.D.); la Asociación Americana de la Diabetes, subvención 1-13-IN-70 (F.D.); el Programa del Proyecto Piloto de la Enfermedad de Alzheimer de la Universidad de California, Davis (F.D.); Instituto Nacional sobre el Envejecimiento, subsidio P30AG010129 (C.D.); y una Subvención “Vision” del Sistema de Salud de la Universidad de California, Davis (F.D.).

**El Centro de la Enfermedad de Alzheimer de UC Davis** es uno de sólo 29 centros de investigación designados por los Institutos Nacionales del Instituto Nacional de Salud sobre el Envejecimiento. El objetivo del centro es traducir los avances de la investigación en mejores diagnósticos y tratamientos para pacientes, concentrándose al mismo tiempo en la meta de largo plazo de encontrar una forma de prevenir o curar la enfermedad de Alzheimer. También financiado por el estado de California, el centro les permite a los investigadores estudiar los efectos de la enfermedad en una población especialmente diversa. Para mayor información, visite [alzheimer.ucdavis.edu](http://alzheimer.ucdavis.edu).

# What Are the Early Warning Signs of Alzheimer's?

by: Dr. Sarah Tomaszewski Farias



An early hallmark feature of Alzheimer's disease (AD) is memory loss. However, the early memory loss associated with AD is of a specific type: it affects the ability to recall recent events or newly learned information (as opposed to memories from a long time ago). Researchers out of the UC Davis Alzheimer's Disease Center have shown that there are a number of everyday

memory problems that are more likely to occur in individuals with Mild Cognitive Impairment (MCI) - often an early stage of AD – as compared to healthy elders.

## These early memory problems can include:

- Difficulty recalling recent conversations
- Problems remembering a short list of shopping items
- Difficulty remembering recent movies or TV shows
- Forgetting appointments, meetings, or other engagements
- Frequently repeating oneself

Changes in other areas of thinking can also occur in early in the course of Alzheimer's. In particular, difficulties with planning, organization, and problem solving – abilities we often refer to as ‘executive functions’ - can also be early warning signs.

## Such problems can present in daily life as:

- Difficulty tracking bills and bank accounts
- Problems planning social events
- Difficulty planning complex meals or following a recipe
- An increased tendency to get side-tracked or distracted during daily tasks
- Difficulty playing games of skill
- Reduced efficiency in prioritizing important tasks

Other cognitive changes that are sometimes associated with early AD include difficulty recalling

words in conversation, calling things by the wrong name, and/or increased difficulty tracking conversations. Interpreting visual images and geographic surroundings can also be affected in early AD and can lead to problems such as difficulty judging distances and recognizing familiar landmarks - both of which can sometimes result in problems with driving.

Early warning signs of AD can also manifest as changes in mood and behavior, including increased depression and nervousness. A decreased tolerance for frustration can also develop leading to irritability. Withdrawing from social activities and hobbies can also occur, in part because of the cognitive demands these activities involve.

It is not uncommon for individuals who are experiencing some of the problems noted above to be unaware that they are having such difficulties. This is most often not caused by psychological ‘denial’ but is a direct manifestation of the disease-related changes occurring in the brain in association with AD. Repeatedly pointing out these problems to the affected person typically does not help them to become aware of the problems, and can lead to increased feelings of frustration. In some sense, it is not surprising that individuals who are having difficulty with memory, can't remember what they forgot!

The early detection of AD is important for a number of reasons. Initiation of treatment may help slow the disease process. Early detection also helps to ensure that affected individuals and their family can adequately plan for future care needs. Finally, with some safety precautions in place, a greater level of autonomy and independence can be preserved. It is also important to keep in mind that not all memory slips and other ‘senior moments’ are signs of early Alzheimer's disease or other serious medical problems. For example, occasionally forgetting names or appointments - but remembering them later are common experiences and are not necessarily cause for alarm. However, if you or a loved one have noticed persistent problems in multiple areas noted above, consider seeking the advice of a doctor.

# *Announcements*

## **UC Davis Alzheimer's Disease Clinic - East Bay New Location in Richmond**

The East Bay clinic of the UC Davis Alzheimer's Disease Center has relocated from Martinez to Richmond. We are very pleased with our new location at 2600 MacDonald Avenue, which we are sharing with LifeLong Medical Care. The clinic is in a lovely home/office building that was once the home of a physician who lived upstairs and shared the offices downstairs with his brother, a dentist. It is directly across the street from the Richmond Civic Center only a few minutes off of Highway 580 and has ample, free parking. Although the clinic has moved, our staff and our services remain the same. We look forward to seeing you there.

Our administrative offices have relocated to the main UC Davis campus. All mail should be directed there. Use the main telephone number for all phone calls. Our new contacts are:

**Send Mail to:**

UC Davis Alzheimer's Disease Center - *East Bay*  
One Shields Avenue - TB171  
University of California  
Davis, CA 95616-5270

**New Clinic Address:**

UC Davis Alzheimer's Disease Clinic - *East Bay*  
2600 MacDonald Avenue  
Richmond, CA 94804

**Main Line:** 1-855-420-2612    **Fax:** 510-752-8937

## **La Clínica de la Enfermedad de Alzheimer de UC Davis - *East Bay* se Mueve a Richmond**

La clínica del Este de la Bahía del Centro de la Enfermedad de Alzheimer de UC Davis se ha transladado de Martinez a Richmond. Estamos muy contentos con nuestra nueva ubicación en el 2600 MacDonald Ave, la cual estamos compartiendo con Life Long Medical Care. La clínica se encuentra en una casa/oficina encantadora, la cual fue la casa de un médico que vivía en la planta superior y compartía las oficinas en la planta baja con su hermano, un dentista. Está directamente ubicada al otro lado de la calle del Centro Cívico de Richmond, apenas unos minutos de la autopista 580 y tiene un amplio aparcamiento gratis. A pesar de que la clínica se ha transladado, nuestro personal y los servicios siguen siendo los mismos. Esperamos verles allí.

Nuestras oficinas administrativas se han reubicado a la ciudad universitaria de UC Davis. Toda correspondencia debe ser dirigida allí. Utilice el número de teléfono principal para todas las llamadas telefónicas.

Nuestra nueva información de contacto es:

**Enviar todo el correo aquí:**

UC Davis Alzheimer's Disease Center – *East Bay*  
One Shields Avenue – TB171  
University of California  
Davis, Ca. 95616-5270

**La clínica:**

UC Davis Alzheimer's Disease Clinic - *East Bay*  
2600 MacDonald Avenue  
Richmond, CA 94804

**Numero principal:** 1-855-420-2612    **Numero de FAX:** 530-752-8937

# Announcements

## Spanish Mini-Medical School UC Davis MIND Institute in Sacramento

### The Spanish Mini Medical School is back!

Health professionals will be giving lectures on a variety of topics related to brain health and aging. This event is organized by the UC Davis Alzheimer's Disease Center, the Latino Aging Research Resource Center, and the Center for Reducing Health Disparities. **The event will be presented in Spanish.**

*Saturday, September 7th, 2013*

*8:30 a.m. - 12:30 p.m.*

**UC Davis MIND Institute**

2825 50th St.

Sacramento, CA 95817

**This event is open to the public but space is limited.**

**Call Rebekha Alfaro at (916) 734-5243 to reserve your spot!**

*Para información en español, página 6*

## UC Davis Alzheimer's Disease Center Research Symposium

This year's schedule will include speakers from the UC Davis Alzheimer's Disease Center, pilot grant recipients, and a poster presentation highlighting AD-related research across the University.

### Research Themes of the Center:

- 1) Modifiable risk factors of late-life cognitive decline
- 2) Biological mechanisms of brain aging and dementia
- 3) Vascular effects on brain structure and function
- 4) Ethnic differences in cognitive aging
- 5) Mixed pathologies
- 6) Markers of early change  
(before Diagnosis of Alzheimer's disease)
- 7) Caregiver support

### Types of Research:

- 1) Observational and clinical studies
- 2) Experimental studies
- 3) Secondary data analysis
- 4) Animal and in vitro models

*Friday, November 1, 2013*  
*11 a.m. - 3 p.m. (lunch included)*  
**UC Davis Conference Center**

Ballrooms AB&C  
550 Alumni Lane,  
Davis, CA 95616

UC Davis Alzheimer's Disease Center (6864)  
4860 Y Street, Suite 3900  
Sacramento, CA 95817

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**Alzheimer's Disease Center**  
<http://alzheimer.ucdavis.edu>

**Latino Aging Research  
Resource Center**  
[www.ucdmc.ucdavis.edu/latinoaging/](http://www.ucdmc.ucdavis.edu/latinoaging/)



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news.**

[www.facebook.com/  
UCDavisAlzheimersDiseaseCenter/](https://www.facebook.com/UCDavisAlzheimersDiseaseCenter/)

## Join Us

*Join us for the following presentations at the MIND Institute  
2825 50th Street, Sacramento, CA*

**The Long Term Consequences of Strokes**

by Dr. Berneet Kaur, UC Davis  
*August 15, 2013, 6 p.m.*

**The 90+ Study**

by Dr. Claudia Kawas, UC Irvine  
*October 24, 2013, 6 p.m.*

**Heart Disease, Diabetes, and Dementia, What  
is the Connection?**

by Dr. Bruce Reed, UC Davis  
*September 5th, 2013, 6 p.m.*

**Alzheimer's Disease Caregiver Workshop**

*November 9, 2013, 8 a.m. - 12 p.m.*

**Promoting Brain Health and Managing  
Cognitive Abilities Among Older Adults**

Dr. Sarah Farias, UC Davis  
*December 5, 2013, 6 p.m.*

UNIVERSITY OF CALIFORNIA, DAVIS ALZHEIMER'S DISEASE CENTER is funded by the National Institute on Aging and the California Department of Public Health.

The UC Davis Alzheimer's Disease Center includes members of the following UC Davis departments: Epidemiology and Preventive Medicine, Neurology, Psychiatry, Internal Medicine and Pathology

**UC Davis ADC - East Bay**  
One Shields Avenue - TB171  
Davis, CA 95616  
Toll-Free: 1-855-420-2612  
FAX (530) 752-8937

**UC Davis ADC - Sacramento**  
4860 Y Street, Suite 3900  
Sacramento, CA 95817  
(916) 734-5496; FAX (916) 703-5290