

Alzheimer's

D I S E A S E C E N T E R N E W S

FROM THE DIRECTOR | *Roger N. Rosenberg, M.D.*



Although Alois Alzheimer described the disease that bears his name in 1906, real biological insights into the disease's cause have become apparent only in the last quarter-century. When I was a medical student 50 years ago, Alzheimer's disease was only briefly mentioned in neuroscience lectures. It was assumed to be caused by atherosclerosis, or "hardening of the arteries."

In the 1980s, several back-to-back discoveries overturned that theory. First, Alzheimer's was associated with increased levels of two proteins: plaques of amyloid protein around nerve cells and tangles of a protein called tau within the cells. Alzheimer's was also found to be related to nerve death and brain atrophy. Finally, all three events – amyloid plaques, tau accumulation and neuronal loss – were definitively linked to progressive memory loss and eventually dementia.

All of these findings were com-

PELLING, but I was always struck by the clustering of the disease in families around the world. Examples were documented in which half the children of a parent with the disease were affected, indicating that a single dominant gene was involved.

This clear evidence of an important genetic role in Alzheimer's disease spurred me to direct my research energies against this devastating and very common neurological disease. I had completed my clinical training in neurology at the Neurological Institute of New York and my training in molecular genetics in the laboratory of Nobel laureate Dr. Marshall Nirenberg at the National Institutes of Health in 1970. I had started doing clinical and biochemical genetic studies on Machado-Joseph disease, a neurodegenerative disease caused by a dominant gene. However, in 1987 I received the funding to establish an NIH-funded Alzheimer's Disease Center here at UT Southwestern Medical Center.

It is a very exciting time to pursue the genetics of Alzheimer's disease. Amyloid-protein genetics has emerged as a key and early event. Three genes have been discovered that cause Alzheimer's disease in families, all of which cause amyloid to be overproduced and stored abnormally in the brain. Subsequently, tau accumulates; inflammation occurs; and eventually there is brain atrophy due to neuronal loss.

Continued on page 7 »

FALL FORUM WILL FOCUS ON PROTEINS BEHIND ALZHEIMER'S

By Aline McKenzie

Dr. Lennart Mucke, professor of neurology and neuroscience at the University of California, San Francisco, and director and senior investigator of the Gladstone Institute of Neurological Disease, will discuss his research on proteins involved in neurodegenerative diseases at a Fall Public Forum on Nov. 3 at UT Southwestern Medical Center.

The abnormal buildup of various proteins, both inside and outside of brain cells, is associated with Alzheimer's and Parkinson's diseases. Dr. Mucke's research focuses on how the proteins contribute to the malfunction of brain cells. Proteins that are harmful on their own can interact with others to cause even greater neurologic damage, he has found.

"We're trying to find out what causes the cognitive impairment

Continued on page 7 »

WHAT'S INSIDE

- Clinical Trials
- Research Studies
- Memory Tips



Research Studies



PART OF THE SOLUTION: HEALTHY VOLUNTEERS TAKE ACTIVE ROLE IN ALZHEIMER'S RESEARCH

By Amanda Siegfried

Joanne Bara, 53, knew there was a family history of Alzheimer's disease on her mother's side. So she wasn't too surprised when her mother was diagnosed with the disease at age 60.

Her father's diagnosis, however, was completely unexpected. The news, said Ms. Bara, came just three months after her mother's death.

Although Ms. Bara is symptom-free, she has no idea whether Alzheimer's might someday affect her. But she's taking no chances.

"When my mother was first diagnosed, I brought her to Dr. Myron Weiner at the Alzheimer's Disease Center for treatment and to participate in drug studies," said Ms. Bara, who at the time worked at UT Southwestern University Hospital-Zale Lipshy. "That's when I became aware that there were also some possibilities to get involved in studies myself."

Ms. Bara, a Carrollton resident, serves as a healthy control subject in an observational study. These types of studies do not require or offer a new treatment, but they are the foundation for future research and therapeutic trials, said Kristin Martin-Cook, clinical research coordinator in the memory research unit of the Alzheimer's Disease Center at UT Southwestern.

Healthy volunteers as well as patients with cognitive impairment, dementia or Alzheimer's are encouraged to participate in these types of studies. The center also has opportunities for volunteers to sign up for trials of the newest experimental drug therapies.

As a volunteer, Ms. Bara visits the center about once a year for



Research study coordinator Kenneth Miles watches study volunteer Joanne Bara complete a memory test. Ms. Bara, who serves as a healthy control subject, says both her parents had Alzheimer's disease.

cognitive tests and to have blood drawn. Data from the tests and blood samples are used in ongoing research studies at UT Southwestern and elsewhere across the state and the country.

"My goal in participating in these tests is that as soon as I see any kind of decline from year to year, in mental status or outcome of the exams, perhaps I can begin treatment earlier than many people do," said Ms. Bara. "I got involved with the hope that if it does happen, I might be able to control it somewhat."

"One of the things I have learned is that you can have Alzheimer's for many years before you actually show clinical symptoms."

For Richard Smith, that day has already arrived.

Although he has not been diag-

nosed with Alzheimer's, Mr. Smith, 89, does suffer from mild cognitive impairment and memory loss. He began volunteering for observational studies at the Alzheimer's Disease Center about seven years ago.

"They gave me memory tests, and mine weren't any good. They also made an MRI – sliced up my brain to see if there were any holes in it," joked Mr. Smith, who lives in Duncanville.

"I learned a lot about memory and memory loss. I enjoyed participating and talking with the people at the center, doing what I could to give them information about how my brain reacted" to the cognitive and memory tests.

"If they can find out something about my memory loss, that might help somebody else, too."

In the meantime, Mr. Smith has a few strategies for coping with his memory loss.

"I make notes all the time," he said. "I'm not too systematic about it, though, because I'll make a note and lose it. But I'm getting better. I'm going to do what I can for as long as I can." *



Richard Smith, who began volunteering for observational studies at the center about seven years ago, works with clinical research coordinator Kristin Martin-Cook.

CLINICAL TRIALS

To learn more about studies under way, call the UT Southwestern Memory Research Unit at 214-648-9376.

DAVUNETIDE

Participants with progressive supranuclear palsy will receive placebo or davunetide nasal spray twice a day for 52 weeks. Participants must be 41 to 85 years old; be able to tolerate magnetic resonance imaging and lumbar punctures; be able to ambulate independently or with limited assistance; have a caregiver willing to attend all study visits and monitor study medication dosing; and have a 12-month history of postural instability or falls. No memory medications are allowed. Call Jackie Rabb, 214-648-9376.

IMMUNE GLOBULIN

Participants with mild to moderate Alzheimer's will receive infusions

of immune globulin, a drug already approved to treat several other conditions. During the 82-week trial, participants will receive 36 intravenous infusions – the first three will be at the clinic and the remainder will be administered by a home health care professional. Call Charlotte Isah, 214-648-9310.

GAMMA SECRETASE INHIBITOR

People with mild cognitive impairment are sought to test a drug that acts against gamma secretase, an enzyme that helps produce beta-amyloid. The study involves 17 to 23 clinic visits over a minimum of two years, with a 50 percent chance of receiving the drug. Participants will undergo physical, neurologic, cognitive and behavioral tests, plus brain imaging and sampling of cerebrospinal fluid. Call Kathy Koch, 214-648-9343.

THE TENT STUDY

People with mild memory loss are sought for a two-month study on whether sleeping in an "altitude tent" and exercising at sea level can boost the levels of protective proteins in the brain and blood. Call Kristin Martin-Cook, 214-648-9368.

EXERCISE STUDY

People with mild cognitive impairment who do not currently adhere to a regular fitness program are sought for a one-year study that will compare individually tailored, supervised aerobic exercise training with flexibility and stretching training. The study will investigate the effects of exercise on cognitive and cardiovascular health as well as changes in proteins known to damage and/or protect the brain. Call Kristin Martin-Cook, 214-648-9368, or Estee Brunk, 214-345-4665.

OBSERVATIONAL STUDIES

While these studies do not require or offer a new treatment, they are the foundation for future research and therapeutic trials.

ADNI-GO (ALZHEIMER'S DISEASE NEUROIMAGING INITIATIVE – GRAND OPPORTUNITY)

This study will build on the first ADNI study with a new group of participants at the earliest stages of mild cognitive impairment. Each patient will undergo magnetic resonance imaging (MRI), traditional positron emission tomography (PET) scanning, new amyloid PET scanning and cerebrospinal fluid analysis, plus standard neuropsychological testing. Call Kristin Martin-Cook, 214-648-9368.

TELECOGNITIVE STUDY

This study compares the feasibility and reliability of doing cognitive testing using a computer monitor or TV screen versus a person-to-person standard test. Participants will undergo two sets of 45-minute tests both in person and by videoconference. A home computer is not necessary, and participants will receive a \$25 Wal-Mart gift card. Call Lara Graham, 214-648-4642.

LATE-ONSET ALZHEIMER'S DISEASE (LOAD) GENETICS INITIATIVE

The goal of this national study is to find genes within a family that may play a role in late-onset Alzheimer's. Participants must include two siblings age 60 or above with Alzheimer's, and at least one other healthy family member age 60 or above. All participants must be able to provide

blood samples and health information. Call Barb Davis, 214-648-9367.

CORE AND TARC RESEARCH STUDIES

People with mild cognitive impairment, early Alzheimer's disease or frontotemporal dementia are always needed for observational studies, as are healthy people 65 or older to serve as controls. The studies usually involve one visit a year for neuropsychological testing, neurological exams, brain imaging and blood sampling. The data collected from these visits is used by many investigators studying the aging brain and disorders of cognitive function. These visits may involve financial compensation. Call Mac Miles, 214-648-9338, Jackie Rabb, 214-648-9376, or Kristin Martin-Cook, 214-648-9368. *

'COWBOY SCIENCE' PAYS OFF WITH DISCOVERY OF KEY MOLECULE

By Aline McKenzie

Sometimes taking a big chance can pay off in a big way. That's what happened to Dr. Steven McKnight, chairman of biochemistry at UT Southwestern, after he received a \$2.5 million National Institutes of Health Director's Pioneer Award.

UT Southwestern researchers led by Dr. McKnight and Dr. Andrew Pieper, assistant professor of psychiatry and biochemistry, have found a compound that preserves newly created brain cells and boosts learning

hippocampus. Humans who lack this gene have a variety of learning disabilities, and the "knockout" rodents show related abnormalities as well as a poorly formed hippocampus. When the "knockout" mouse received P7C3, however, normal structure and function of the hippocampus were restored.

In elderly rats, which characteristically show a decline in the birth and formation of hippocampal neurons, the researchers found that

on something safe – I'm going to go for it. That's what the NIH expected of me and my team," said Dr. McKnight, who holds the Distinguished Chair in Basic Biomedical Research and the Sam G. Winstead and F. Andrew Bell Distinguished Chair in Biochemistry. "I'd like to give the NIH credit for betting on 'cowboy' science. If this pans out, it will be the most useful contribution of my career."

Dr. Francis Collins, director of the NIH, said Dr. McKnight's results exactly fit the award's purpose.

"The NIH Director's Pioneer Award gives highly innovative investigators the freedom to pursue bold new avenues of research. Such approaches can yield substantial payoffs, as in the case of the exciting clinical implications of Professor McKnight's basic neurobiological research discovery," Dr. Collins said.

Other UT Southwestern researchers involved in the study were Dr. Jeffrey Long and Shanhai Xie, both research scientists in biochemistry; graduate students Ginger Becker, Emanuela Capota, Ching-Han Shen and Kerstin Ure; Sandi Jo Estill, research associate in biochemistry; Jeannie Zhong, research scientist in psychiatry; Paula Huntington, research associate in psychiatry; medical student Shauna Goldman; Maria Capota and Jeremiah Britt, both research technicians in psychiatry; Dr. Tiina Kotti, assistant professor of molecular genetics; Dr. Noelle Williams, associate professor of biochemistry; Dr. Karen MacMillan, assistant instructor of biochemistry; Jacinth Naidoo, research assistant in biochemistry; Lisa Melito, research associate in biochemistry; Dr. Jenny Hsieh, assistant professor of molecular biology; Dr. Jef De Brabander, professor of biochemistry; and Dr. Joseph Ready, associate professor of biochemistry.

Dr. Daniel Brat of Emory University School of Medicine also participated in the study. *



Drs. Joseph Ready (from left), Jef De Brabander, Andrew Pieper and Steven McKnight discovered a compound that boosts learning and memory in rodents.

and memory in an animal study that appears in *Cell*.

Over a three-year period, the research screened 1,000 individual molecules to see which ones might enhance the production of neurons in the adult mouse hippocampus, a region of the brain critical to learning and memory. The scientists found that one of the compounds, called P7C3, achieved this by protecting newborn neurons from dying.

The researchers then administered P7C3 to "knockout" mice lacking a gene that controls the generation of new neurons in the

P7C3 increased both the birth and survival of new neurons, and the memory and learning capability of the aged rats.

"It's been a wonderful experience," Dr. Pieper said. "At first there was a lot of doubt, because we could have gone through the whole screen and found nothing."

Dr. McKnight was one of the first 12 recipients of the NIH Director's Pioneer Award, which is designed to allow researchers to pursue risky experiments that have the potential for producing highly innovative results.

"When I received the award, I thought 'I'm not going to waste it

RESEARCHERS FIND KEY TO HOW MOLECULE RAISES ALZHEIMER'S RISK

By Aline McKenzie

One in every six people carries a gene called APOE4. Those with the gene have up to 10 times the risk of developing Alzheimer's disease at an early age.

Now UT Southwestern researchers are closer to figuring out how the molecule APOE4, which the gene produces, acts to interfere with the normal function of brain cells.

The researchers discovered that APOE4 makes a nerve cell hold back the molecules that enables it to respond to other cells, thereby disabling a chemical process known to be important in learning. Their findings appear online in the *Proceedings of the National Academy of Sciences*.

"This is actually a fairly simple system," said Dr. Joachim Herz, director of the Center for Alzheimer's and Neurodegenerative Diseases at UT Southwestern and senior author of the study. "For the first time, we see an uninterrupted biochemical pathway that links the surface of the brain cell to the dysfunction inside the cell, and specifically at the junction at which nerve cells communicate."

"The question is, now that we've apparently identified what's going on, can we do anything about this disease process at the fundamental molecular level? That's what we're working on right now."

- Joachim Herz, M.D.

The research focused on a basic characteristic of nerve cells called neurotransmission, in which they use chemicals to signal each other.

When one nerve cell needs to "talk" to another, its tip sends out

a chemical called a neurotransmitter. The surface of the second cell is studded with molecules called receptors, which fit specific neurotransmitters like a lock and key. When a neurotransmitter docks onto its receptor, the second cell responds.

A cell can fine-tune its sensitivity by removing receptors from its surface. It can eventually recycle them back to the surface, where they can respond to neurotransmitters again.

signals; however, the normal mice could recover from this suppression, while the mice with APOE4 could not.

Knowing how a biological system works doesn't automatically translate to clinical use, cautioned Dr. Herz, who holds the Thomas O. and Cinda Hicks Family Distinguished Chair in Alzheimer's Disease Research. "Although these findings constitute a milestone in our understanding of how APOE4 becomes



Dr. Joachim Herz recently bicycled from Dallas to Oklahoma as part of the Alzheimer's Breakthrough Ride to raise funding and awareness for Alzheimer's disease research.

The researchers looked at receptors that respond to a neurotransmitter called glutamate, which is implicated in memory and learning. In mice that were genetically altered to make human APOE4, the researchers found that APOE4 prevented the cells from accomplishing a vital step in learning – becoming more sensitive to repeated signals.

The researchers also studied the mice's hippocampus – an area of the brain vital to learning – to see how it would respond to extracts from the brain of a human with Alzheimer's. The extract prevented both normal and genetically altered mice from processing incoming

such a potent risk factor for Alzheimer's disease, potential drugs that might come from this finding would still require years of development," he said.

"The question is, now that we've apparently identified what's going on, can we do anything about this disease process at the fundamental molecular level? That's what we're working on right now."

Other UT Southwestern researchers involved in the study were graduate student Ying Chen; Dr. Murat Durakoglugil, assistant instructor of molecular genetics; and Dr. Xunde Xian, postdoctoral researcher in molecular genetics. *

REDUCING ALZHEIMER’S-RELATED PROTEIN SHOWS PROMISE FOR KIDS WITH DOWN SYNDROME

By Aline McKenzie

Much Alzheimer’s research focuses on a protein called beta-amyloid. That same protein may also hold the key to helping children with Down syndrome.

Researchers at UT Southwestern have found that reducing beta-amyloid in young mice with a condition resembling Down syndrome improves their ability to learn.

“This preliminary study in the animal model raises the intriguing possibility that drugs that lower beta-amyloid levels might offer some benefit to children with Down syndrome,” said Dr. Craig Powell, assistant professor of neurology at UT Southwestern and co-lead author of the study, which is available in *PLoS One*, the Public Library of Science’s online journal.

Down syndrome, a genetic disease that causes learning disabilities and physical problems, is caused by an extra copy of chromosome 21. This chromosome includes the genes for proteins that produce beta-amyloid, a protein that accumulates in the brains of people with Alzheimer’s disease and is believed to contribute to cognitive decline.

Children with Down syndrome have increased normal levels of beta-amyloid in their brains, but it is unknown whether the increased levels affect intellectual abilities, Dr. Powell said. By age 40, nearly all adults with Down syndrome develop signs of Alzheimer’s, with dementia developing in their 50s and 60s.

For the study, the researchers used mice with a genetic anomaly that closely mimics human Down syndrome. These mice have three copies of a stretch of genes, including those related to beta-amyloid production, and also display learning disabilities, including difficulties learning a standard water maze.

The scientists treated 4-month-old genetically altered mice with DAPT, an experimental drug that blocks gamma-secretase, an enzyme essential for beta-amyloid production. A four-day treatment lowered beta-amyloid levels by 40 percent and significantly improved the rodents’ performance to the point that they learned the maze as quickly as normal mice.

Dr. Powell, however, cautioned that the blocked enzyme is involved in

many brain functions besides creating beta-amyloid.

“Current gamma-secretase inhibitors may have untoward side effects,” he said. “The goal now is to identify drugs that block the ability of gamma-secretase to create amyloid without blocking its ability to perform its other tasks.”

Dr. Jacqueline Blundell, former postdoctoral fellow at UT Southwestern, also participated in the study, as did researchers from The Rockefeller University and Columbia University Medical Center.

The study was funded by the Lowe Foundation, the Crystal Charity Ball, the Van Beber family, the David M. Crowley Foundation, the National Institutes of Health, The Fisher Center for Alzheimer’s Disease Research Foundation and the F.M. Kirby Foundation. *



Craig Powell, M.D., Ph.D.

FRIENDS GRANT HELPS RESEARCHERS PURSUE ALZHEIMER’S VACCINE

The Friends of the Alzheimer’s Disease Center provide research grants to UT Southwestern faculty to develop new insights into the disease that might lead to new therapies. This year, the Friends awarded a \$65,000 grant to Dr. Todd N. Eagar, assistant professor of pathology and immunology.

Dr. Eagar’s research, in collaboration with Dr. Roger Rosenberg, centers on the interactions between the immune and central nervous systems, with the goal of developing a vaccine against amyloid plaques, or clusters, in the brains of patients

with Alzheimer’s disease.

Researchers elsewhere have performed clinical trials of a vaccine using beta-amyloid, the protein that makes up the plaques. Although this vaccine reduced the amount of plaques found in the brain, several subjects developed a form of encephalitis, or brain inflammation, caused by cells in the immune system. Dr. Eagar is exploring the use of a novel gene vaccine that uses DNA instead of the protein itself.

Previous work using a mouse model for Alzheimer’s disease has shown that the gene vaccine can

reduce the levels of amyloid plaque without generating the strong cellular immune response that could cause encephalitis.

The research supported by this grant will allow Dr Eagar’s lab to characterize the immune response generated by the gene vaccine and to improve the vaccine’s effectiveness and safety. *



Todd N. Eagar, Ph.D.

FROM THE DIRECTOR

Continued from page 1

We and others around the world have searched for and found many genes that cause these abnormal protein accumulations by scanning the DNA of many people to find genetic variations associated with Alzheimer’s. This effort has been successful, and we are now investigating what functions are being disturbed and what potential new therapies might be developed.

I have set a goal to use genetic strategies to prevent or even cure Alzheimer’s disease. For the past seven years, I have been working with my colleagues Drs. Bao-Xi Qu, Doris Lambracht-Washington, Todd Eagar

and Olaf Stüve to do just that.

In the past four years, we have published papers in the *Journal of the Neurological Sciences*, *The Journal of the American Medical Association* and *Vaccine* showing that a DNA vaccine can more than halve brain levels of the toxic amyloid protein in animal studies. The immune response produced by the vaccine is unlikely to cause side effects. If this result could be obtained in patients with Alzheimer’s disease, it might delay the onset of disease by five years and reduce its prevalence by 50 percent.

Our efforts now are directed at

obtaining Food and Drug Administration approval and NIH funding for a clinical trial. We hope to begin within two years.

Over the last 22 years, I have been most fortunate to work with dedicated and creative research and clinical colleagues to initiate and sustain this program in genetic research. We all hope that the DNA vaccine, which is the outgrowth of many years of basic science research, will reach the clinic and will offer hope and benefit to our patients. *

Dr. Rosenberg holds the Abe (Brunky), Morris and William Zale Distinguished Chair in Neurology.

FALL FORUM FOCUSES ON PROTEINS BEHIND ALZHEIMER’S

Continued from page 1

and discover ways to make the brain more resistant to these damaging proteins,” said Dr. Mucke, who received this year’s Potamkin Prize for Research in Pick’s, Alzheimer’s, and Related Diseases, awarded by the American Academy of Neurology.

“If one could remove the toxic proteins from the brain or block their detrimental actions, I can imagine that there might be a remarkable recovery of function.”

The 7 p.m. forum, presented by the Friends of the Alzheimer’s Disease Center at UT Southwestern, is titled “What Will It Take to Defeat Alzheimer’s Disease?” It will be held in the Simmons/Hamon Biomedical Research Buildings on the North Campus, 6000 Harry Hines Blvd. Complimentary valet parking is available.

The forum is free to the public, but because seating is limited, attendance should be confirmed by calling the medical center’s Office of Development at 214-648-2344.

Alzheimer’s disease is associated with the abnormal accumulation of several proteins; beta-amyloid and tau are the most infamous. Beta-amyloid forms clusters called plaques outside the brain cells of people

with the disease. Tau protein, which normally stabilizes the shape of brain cells, creates abnormal clumps within the cells during the disease. Parkinson’s disease is associated with another protein, alpha-synuclein, which forms clumps in the brain called Lewy bodies.

Dr. Mucke found that beta-amyloid can damage connections between nerve cells even without being deposited into visible plaques. “These plaques are like large garbage dumps of amyloid,” he said. “We think they’re relatively inert compared to these cruise missiles that are floating around and that impair the communications between brain cells.”

Using mice that have been genetically engineered to model both Alzheimer’s and Parkinson’s diseases, Dr. Mucke and his colleagues have found that beta-amyloid and alpha-synuclein can interact to cause even worse cognitive and motor problems than either one alone.

Dr. Mucke also discovered that in mouse models of Alzheimer’s disease, brain activity alternates between an epilepsy-like abnormal excitation and abnormal inhibition. Using genetic engineering to reduce the amount of tau protein in mice

prevented these alterations and halted cognitive deficits induced by beta-amyloid.

“If drugs that reduce tau could be found, this approach could complement strategies aimed at beta-amyloid itself, and might prove especially useful if the efficacy and long-term safety of anti-amyloid strategies turn out to be limited,” he said.

In ongoing studies, Dr. Mucke and his colleagues are exploring how beta-amyloid affects nerve function and survival. They are also looking at how reducing tau protein protects against deficits induced by beta-amyloid.

The Friends of the Alzheimer’s Disease Center was established in 1996 to provide financial support for Alzheimer’s research at UT Southwestern. All of the group’s contributions go directly to support Alzheimer’s research at the medical center.

Since its founding, the group has raised \$989,236 for grants to researchers. For information on joining the group, call the Office of Development at 214-648-2344. *



Lennart Mucke, M.D.

Join Us



JOIN THE FRIENDS OF THE ALZHEIMER'S DISEASE CENTER

The Friends of the Alzheimer's Disease Center raises funds to establish yearly grants for promising researchers who are working to unlock the mysteries of Alzheimer's disease. In addition, the Friends sponsor public forums each spring and fall to present the latest scientific information on the disease to the general public. We welcome the community to these sessions.

Fully tax-deductible memberships in the Friends begin at \$500 per year per individual or couple. To join the Friends, call 214-648-2344 or visit our website at www.utsouthwestern.edu/donatenow. Honorary or memorial gifts are another meaningful way to make contributions toward research that is under way at UT Southwestern.

The Alzheimer's Disease Center News is published by The University of Texas Southwestern Medical Center at Dallas. We welcome your comments or suggestions at 214-648-3404 or robin.loveman@utsouthwestern.edu

Director – Dr. Roger N. Rosenberg

Editor – Robin P. Loveman

Writers – Aline McKenzie, Amanda Siegfried

STRATEGIES CAN HELP EASE EFFECTS OF MILD MEMORY LOSS

“Senior moments” may be the stuff of jokes, but for people with mild cognitive impairment or Alzheimer's disease, memory loss is a frustrating reality. Some simple strategies can help, however.

“Small changes in everyday life can minimize the impact of memory problems,” says Kristin Martin-Cook, clinical research coordinator for the Alzheimer's Disease Center at UT Southwestern Medical Center.

Ms. Martin-Cook suggests some of the following memory aids:

- Use medication dispensers, whether something as simple as a plastic pill organizer or as complex as an electronic dispenser with alarms.
- Keep a calendar, with enough details that can cue memory, in a prominent location.
- Keep a summary of medical history, current medications and allergies, emergency contacts, and doctors' phone numbers on the refrigerator at home and in one's wallet.
- Register with the Alzheimer's Association's Safe Return program, which provides a medical alert bracelet.
- Rely on routine and familiarity. “New learning is difficult when short-term memory is a weakness, so stick with the same phone, same car, same appliances whenever possible,” Ms. Martin-Cook says.

Nonprofit Org.
U.S. POSTAGE
PAID
Dallas, Texas
Permit No. 4994

Alzheimer's Disease Center
UT SOUTHWESTERN
MEDICAL CENTER
5323 Harry Hines Blvd.
Dallas, Texas 75390-9009
ADDRESS SERVICE REQUESTED