Volume 9, Number 4 Alzheimer's Disease Education & Referral Center

CONNECTIONS

Frontotemporal Dementia: Growing Interest in a Rare Dementia

Editor's Note: This issue of *Connections* focuses on a group of rare diseases called frontotemporal dementia (FTD). These diseases are characterized by an accumulation in the brain of abnormal forms of *tau* protein. Because the causes of dementia can often be misdiagnosed, we are including a chart comparing FTD and Alzheimer's disease (AD), and a chart of certain clinical characteristics of select FTD. We take a closer look at the mechanisms of this class of diseases in the article "Tauopathies: New Discoveries, New Knowledge."

Dementia is frequently equated with Alzheimer's disease. While it is true that AD is the most common cause of dementia, scientists and clinicians have identified a number of additional progressive neurodegenerative conditions that cause dementia, including vascular dementia, dementia with Lewy bodies, and a much rarer group of diseases called frontotemporal dementia. Scientists are showing growing interest in understanding the similarities and differences among these neurodegenerative diseases. Though much is still unknown, the more we learn about these diseases, the more patients and families will benefit. New knowledge about these rarer forms of dementia also may shed light on AD.

What is FTD?

FTD primarily affects the frontal and anterior temporal lobes of the brain. These areas control "executive

(Continued on page 2)

Tauopathies: New Discoveries, New Knowledge

A central characteristic of many neurodegenerative diseases is aggregation of abnormal proteins in the brain. For example, a protein called alpha-synuclein is known to accumulate in Parkinson's disease, and a protein called huntingtin accumulates in Huntington's disease. These abnormal proteins likely play a key role in the dysfunction and death of neurons and the resulting clinical characteristics of the diseases. In the last couple of decades, considerable attention has been focused on one such abnormal protein—beta-amyloid—and the resulting plaques that may lead to AD.

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News From the ADEAR Center
Alzheimer's Disease Education & Referral Center
A Service of the National Institute on Aging

Alzheimer's Disease Cooperative Study

AD Cooperative Study Launches "Healthy Aging & Memory"

The Healthy Aging & Memory Study, a new Alzheimer's Disease Cooperative Study sponsored by the University of California, San Diego and funded by the National Institute on Aging, began recruiting volunteers at 40 sites nationwide in February 2002. The study will assess new scientific tools and methods for measuring and tracking memory changes in healthy seniors. If proven effective, these instruments may help speed clinical trials of promising treatments to prevent AD.

"Now that researchers are moving quickly toward testing treatments that may actually delay or prevent AD, we need to be ready with simple, cost-effective assessment instruments," said Leon Thal, MD, Principal Investigator for the Alzheimer's Disease Cooperative Study. "We hope that the tools we are testing, which minimize time requirements for clinical staff and study participants, will make large-scale AD prevention trials more feasible."

The Healthy Aging & Memory Study will evaluate how reliable, efficient, accurate, and sensitive several new instruments are in detecting changes in normal aging that lead to early AD.

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The lobes of the cerebral cortex: frontal, parietal, temporal, and occipital

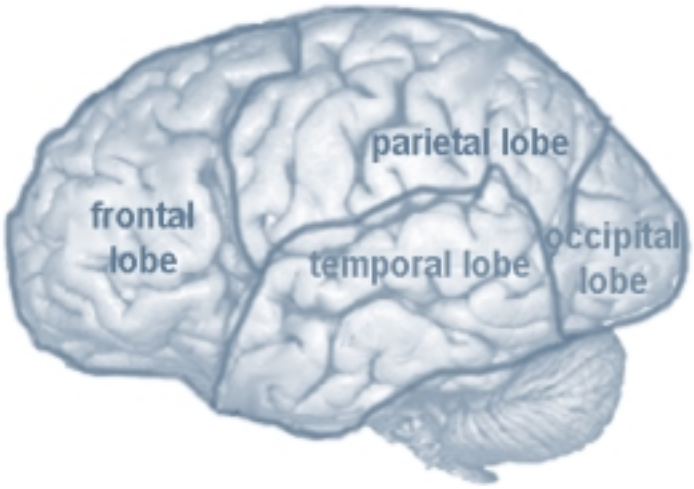

Image courtesy of the University of Texas at Austin, Addiction Science Research & Education Center

What is FTD?

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functions" such as reasoning, personality, social behavior, movement, speech, language, and certain aspects of memory. FTD usually develops between the ages of 35 and 75, and affects men and women about equally. Patients generally live with the disease for 2 to 10 years after diagnosis. FTD appears to be quite rare; researchers believe that FTD accounts for perhaps 3 percent of all dementia cases. The disease appears to have a strong genetic component, for in 20-40 percent of the cases, the person had a family history of dementia. A major scientific breakthrough occurred in 1998 with the discovery that a mutation in the *tau* gene causes a form of FTD called frontotemporal dementia with parkinsonism linked to chromosome 17 (FTDP-17) (see article on page 1 on tauopathies for more on the genetic links to FTD).

What are the Clinical Characteristics of FTD?

FTD is characterized by a gradual onset of changes in personality, social behavior, and language ability. Particular changes depend on whether the damage has primarily affected the right or left side of the front of the brain (language deficits predominate when the left side is primarily affected; behavioral problems develop with right-sided disease). Symptoms include one or more of the following:

- uninhibited and socially inappropriate behaviors (people begin to do things they never would before, such as stealing or drinking out of the punch bowl at a party)
- inappropriate sexual behavior
- loss of awareness of or concern about the changes in behaviors
- loss of concern about personal appearance and hygiene
- major increase in appetite that leads to constant eating and weight gain
- apathy, loss of drive, social withdrawal, lack of concern and empathy for others
- loss of speech and language (many become completely mute by the middle to late stages)
- compulsive or repetitive behaviors, such as pacing, collecting things, or handwashing
- oral fixation (people begin to put objects as well as food into their mouths); this occurs more in the late stages
- memory loss, although this is not one of the first signs and is less severe relative to other symptoms

People with FTD also have motor difficulties like those seen in Parkinson's. These include rigidity, lack of balance, and stiffness of movement, but not the trembling of arms and legs at rest that are characteristic of Parkinson's. One major difference between FTD and AD is that people with FTD do not suffer memory loss to the same degree as those with AD. People with FTD also remain oriented to time and place and are able to recall information about the past and present. Even in the late stages, people with FTD, unlike those with AD, are able to negotiate their surroundings and know where they are.

What Pathologic Changes Can Occur in FTD?

Though scientists don't yet know the cause of the destructive changes of FTD, they have a fairly good understanding of the changes themselves. One of the most characteristic pathologic changes is an accumulation of abnormal *tau* inside nerve cells in the brain. *Tau* is a protein that plays an essential role by supporting the internal structure of the nerve cell and serving as a part of the transport mechanism for nutrients and other molecules. In FTD, *tau* becomes abnormal and aggregates into tangles. This disrupts normal nerve cell processes and ultimately leads to the death of the cells (see the article on tauopathies for more on the process). Other changes include a progressive loss of nerve cells in the frontal and temporal regions of the brain. Gliosis, a form of tissue scarring in the central nervous system, also occurs, as does vacuolation, a process in which "holes" form in the outer layer of the brain. In Pick's disease, Pick bodies, which are

abnormal cell inclusions, begin to form in the brain.

How is FTD Diagnosed?

Because of its symptoms, FTD is often misdiagnosed as a psychiatric problem or as AD (see chart below for a summary of similarities and differences between AD and FTD). However, a trained and experienced health care professional can look for features that rule out other diagnoses as well as those that pinpoint FTD. Diagnosis generally involves:

- a careful medical history and examination of behavioral changes—because patients lack the ability to recognize changes that have occurred or that their current behavior is problematic, information from family members and others close to the patient is crucial;
- neuropsychological examination, which helps assess language, memory, executive functioning, visual-spatial skills; and
- neuroimaging to determine where and how extensively brain regions have atrophied.

What Treatments are Available for FTD?

Currently, no treatments are available to slow or stop the disease process. However, available psychiatric medications can be used to treat some of the behavioral problems. For example, selective serotonin reuptake inhibitors (SSRIs) or small doses of newer antipsychotic medications, can help to alleviate some of the more difficult behavioral symptoms. Practical strategies can blunt the impact of behavioral symptoms. These strategies include making sure that the person with FTD does not drive and avoids situations that call for financial decisions or those in which judgment is important.

In treating FTD, an accurate diagnosis is crucial. Some medications commonly used to treat other dementias, such as cholinergic drugs and antipsychotic drugs that contain dopamine blockers, are ineffective or could be harmful in a person with FTD. As with other types of progressive neurodegenerative diseases, caring for people with FTD is stressful and challenging. Therefore, providing caregivers with educational materials and sources of support is critical.

The Outlook

Scientists are still grappling with many unanswered questions about frontotemporal dementia and other dementias. They are actively researching all aspects of these disorders to better understand the causes and risk factors. It is hoped that with a better understanding of abnormal *tau* formations, more effective methods of diagnosis and treatment will be found.

Frontotemporal Dementia and Alzheimer's Disease: Similarities and Differences

Features	Frontotemporal Dementia	Alzheimer's Disease
age at which disease generally occurs	♦ usually after age 40 and before 65	♦ usually after 65
brain areas affected	♦ frontal and temporal lobes	♦ starts in the medial temporal area, usually in the hippocampus ♦ spreads to other areas of the brain
pathologic features	♦ loss of nerve cells ♦ no amyloid plaques ♦ *tau* tangles seen in certain FTDs	♦ loss of nerve cells ♦ amyloid plaques ♦ *tau* tangles
clinical features	♦ begins with personality and behavior changes; some may be hyperactive while others seem apathetic ♦ loss of empathy toward others; lack of insight into proper social conduct ♦ memory is preserved early on ♦ language difficulty ♦ compulsive eating and oral fixations ♦ repetitive actions ♦ later in the disease, loss of motor skills, speech and muscle movement	♦ begins with memory loss ♦ patients lose ability to learn new information ♦ patients become unable to orient themselves to time and place ♦ later, personality and behavior problems develop ♦ possible hallucinations and delusions in later stages

Tauopathies

(Continued from page 1)

Within the last few years more attention has been focused on the other hallmark feature of AD—the "tangles" of abnormal *tau* protein. We are learning more about *tau* and what happens to change it in the AD process. Scientists are discovering that these *tau* tangles, along with extensive neuron loss and gliosis (a kind of tissue scarring in the brain), are the major feature of a number of neurodegenerative diseases. A consensus is emerging that these diseases should be called "tauopathies." Tauopathies include frontotemporal dementia (see related article on FTD), Pick's disease, corticobasal degeneration, familial FTDP-17, progressive supranuclear palsy, amyotrophic lateral sclerosis/parkinsonism-dementia complex of Guam, and motor neuron disease (see chart on page 5).

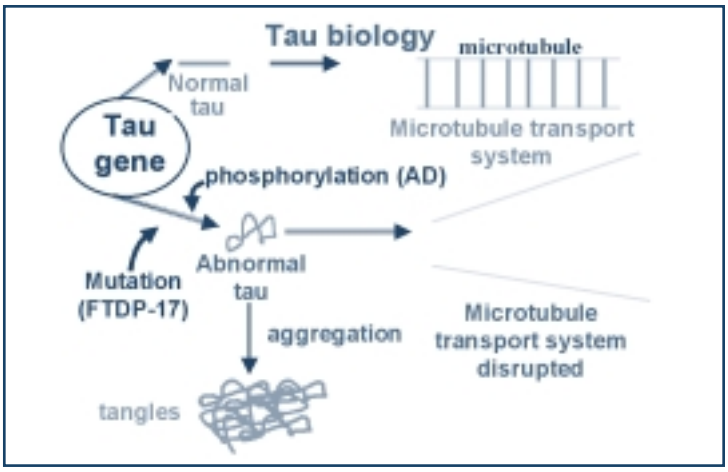
Tau's Vital Role

Tau is a protein commonly found throughout the central nervous system. Six forms of *tau* are found in the adult human brain and each is maintained in a constant proportion. *Tau* binds and helps stabilize microtubules, a component of the nerve cell's internal support structure or skeleton. In the healthy cell, microtubules form structures like train tracks, which guide nutrients and other molecules from the cell body down through the axon. *Tau*, therefore, plays a vital role in ensuring a nerve cell's survival by supporting its structure and facilitating transport.

What Goes Wrong?

Tau normally undergoes a process called phosphorylation in which molecules called phosphates are added to the protein. Scientists have found that *tau* in the tangles that characterize AD and other neurodegenerative diseases is composed of overly and abnormally phosphorylated *tau*. They speculate that this process causes the *tau* to aggregate into tangled filaments instead of attaching itself to the microtubules, thereby destabilizing the microtubules.

Starting in 1998, scientists took a major step forward when they found that mutations in the gene that directs the production of *tau* cause one particular tauopathy—frontotemporal dementia with parkinsonism linked to chromosome 17, or FTDP-17. These mutations appear to lead to problems in two ways—by forming mutant *tau* proteins in cells or by changing the proportion of the forms of *tau* normally expressed in the brain. These changes promote *tau* aggregation into filaments and harm the ability of *tau* to bind to microtubules.



source: Neuroscience and Neuropsychology of Aging, National Institute on Aging

Scientists have a growing body of clues, evidence, and facts about *tau* tangles and what goes wrong. The puzzle is that this pathologic process gives rise to such a wide range of disorders and diseases. These diseases share a number of characteristics, but they also each have distinct features that set them apart from each other, and from AD. This suggests to scientists that environmental and other genetic factors also must play a role in the cause and development of tauopathic conditions.

Transgenic Mouse Models

In 1999, scientists produced several transgenic mouse models that express *tau* tangles. These animals are not complete models of tauopathies, but they will allow researchers to explore normal *tau* function more fully and to study and measure the accumulation of abnormal *tau* deposits in the brain. The models enable investigators to move ahead on several genetic fronts, including determining how known mutations lead to the death of neurons and studying how other mutations may be involved.

Questions Persist

Investigators hope that growing knowledge about *tau* will answer questions about tauopathies as well as lead to further insights about Alzheimer's disease. They want to know, for example:

- What roles do environmental and other genetic factors play in the development of tauopathies, and how do they contribute to the clinical characteristics of these diseases?
- In what different ways does the abnormal *tau* expressed in AD and tauopathies contribute to the development of these diseases?
- Which feature of AD is responsible for the dysfunction and death of neurons—beta-amyloid plaques, or *tau*-containing tangles? If they both play a role, what are those roles and how might they interact? How is the process of plaque formation related to tangle formation?

Progress is being made, however, giving rise to hopes that someday we will have effective treatments and perhaps even preventive measures for these devastating diseases.

Disease	Clinical Characteristics
Pick's Disease	♦ personality and behavioral changes: disinhibition, inappropriate social behaviors, loss of mental flexibility and empathy; development of obsessive-compulsive behaviors, compulsive overeating, food cravings, putting any object in mouth ♦ language problems: use of wrong words, echoing what others say, mutism can develop ♦ difficulties in thinking, concentrating, paying attention; gradual emotional apathy, loss of moral judgment; generalized dementia
FTDP-17	♦ behavioral changes: loss of initiative, disinhibition, obsessive-compulsive behavior, restlessness, verbal aggressiveness ♦ psychiatric symptoms: delusions, visual or auditory hallucinations ♦ cognitive decline: word finding difficulties, other language difficulties though comprehension remains preserved; executive functions, attention, and abstract reasoning become impaired; mutism eventually develops
Supranuclear Palsy	♦ motor difficulties: problems with balance and gait; problems controlling eye movement, involuntary closing of the eyes, inability to maintain eye contact with others; difficulties with swallowing ♦ personality/behavioral changes: apathy, increased irritability, angry outbursts, depression, progressive dementia
Corticobasal Degeneration	♦ signs of parkinsonism: poor coordination, rigidity, impaired balance ♦ cognitive and visual-spatial impairments, loss of ability to make familiar and purposeful movements ♦ hesitant and halting speech ♦ sudden contractions of muscles or muscle groups ♦ difficulty swallowing

Recent Paper Classifies Criteria for FTD

An international team of scientists is proposing guidelines for classifying diseases with certain characteristics under the collective, umbrella term, frontotemporal dementia. Writing in the November 2001 issue of the *Archives of Neurology*, well-known dementia disease researchers Guy M. McKhann, M.D., Marilyn S. Albert, Ph.D., Murray Grossman, M.D., Ph.D., Bruce Miller, M.D., Dennis W. Dickson, M.D., and John Q. Trojanowski, M.D., Ph.D., present a summary of conclusions reached by a larger group of experts on proposed clinical and neuropathological criteria for FTD.

To help general physicians, neurologists, neuropathologists, and psychiatrists understand the heterogenous diseases that make up FTD, the group proposes 5 distinct neuropathological categories, based on presence of abnormal *tau* inclusions, neuron loss and gliosis, 3R *tau* and/or 4R *tau*. Among the diseases which the group recommends classifying as FTD are Pick's disease, FTD with parkinsonism linked to chromosome 17, corticobasal degeneration, progressive supranuclear palsy, and neurofibrillary tangle dementia,

This paper clarifies the demographics of FTD; the core clinical phenotypes of FTD, including the behavioral and language presentation of FTD; and imaging and laboratory data in FTD.

Because these diseases are often misdiagnosed as a psychiatric disorder, and many general physicians believe almost all dementia is AD, the group hopes that this classification system will promote greater understanding in the medical community. The authors hope to improve early and accurate diagnosis of FTD and to continue research to discover effective treatment strategies.

Reference: McKhann et. al. (2001) Clinical and Pathological Diagnosis of Frontotemporal Dementia: Report of the Work Group on Frontotemporal Dementia and Pick's Disease. *Archives of Neurology*, Nov., 58(11), 1803-1809.

See the next page for sources of additional information on FTD, tauopathies, and other dementias.

For More Information on FTD

- Alzheimer's Association newsletter, *Advances*: Frontotemporal Dementia (Summer 2001). Internet: www.alz.org/caregiver/programs/advances.
- Family Caregiver Alliance fact sheet: *Frontotemporal Dementia*. (May 2000). Internet: www.caregiver.org/factsheets/frontotemp.html.
- Family Caregiver Alliance interview with Dr. Bruce Miller. Internet: www.caregiver.org/interviews/bmiller.html.
- John Douglas French Center for Alzheimer's Disease. (1999/2000). Once Confused with Alzheimer's, Frontotemporal Dementia Strikes Patients Earlier, Causing Changes in Communication, Cognition and Personality. *John Douglas French Center Journal*, 10, 2-5. Also available on the Pick's Disease Support Group Web site at www.pdsg.org.uk/articles/JDFC-1.htm.
- Lee, V.M-Y.; Goedert, M.; Trojanowski, J.Q. (2001). Neurodegenerative Tauopathies. *Annual Reviews of Neuroscience*, 24, 1121-1159.
- Miller, B.L. (2001). Tau Mutations — Center Tent or Sideshow? *Archives of Neurology* [Editorial], 58(3), 351-352.
- National Institute of Neurological Disorders and Stroke fact sheets on Pick's disease, dementia with Lewy bodies, progressive supranuclear palsy, and corticobasal degeneration. Internet: www.ninds.nih.gov/health_and_medical/disorder_index/htm.
- Neary, D.; Snowden, J.S.; Mann, D.M.A. (2000). Classification and Description of Frontotemporal Dementias. *Annals of the New York Academy of Sciences*, 920, 46-51.
- Perry, R.J., Miller, B.L. (2001) Behavior and Treatment in Frontotemporal Dementia. *Neurology*, 56(11 Suppl 4), S46-S51.
- Spillantini, M.G.; van Swieten, J.C.; Goedert, M. (2000). *Tau* Gene Mutations in Frontotemporal Dementia and Parkinsonism Linked to Chromosome 17 (FTDP-17). *Neurogenetics*, 2(4), 193-205.

Breakthrough Mouse Model for Alzheimer's More Like Human Disease

In a breakthrough with important implications for research on AD, scientists at the Mayo Clinic Jacksonville (FL) have developed a new mouse model that more closely resembles AD as it appears in humans. The new "double transgenic" mouse is the first to include both the brain plaques and tangles associated with AD. It is expected to contribute considerably to knowledge about the course of the disease and will help in developing and testing therapies.

The research, supported by the National Institute on Aging (NIA) and the National Institute of Neurological Disorders and Stroke, is reported in the August 24, 2001, issue of *Science* by Michael Hutton, Ph.D., Dennis Dickson, M.D., Jada Lewis, Ph.D., Shu-Hui Yen, Ph.D., and Eileen McGowan, Ph.D. of Mayo Jacksonville.

"The development of this type of animal model for Alzheimer's disease is critical to our success in designing effective therapies for tangles and cell death," says Stephen Snyder, Ph.D., Etiology of Alzheimer's Disease program, NIA. "This is a major step, one that I expect can help move us forward greatly in our understanding of AD."

The model was developed by crossbreeding mice with genetic mutations in the proteins associated with plaques and tangles—the *tau* protein involved in neurofibrillary tangles and the amyloid precursor protein (APP) involved in development of amyloid plaques. The new strain of mice, called TAPP (for *tau*/APP), shows evidence of intensified *tau*-related tangles in regions of the brain vulnerable in AD, a finding that may help scientists explain the so-far elusive connection between amyloid pathology and tangle formation.

Expansion Planned for National Cell Repository

Geneticists speculate that as many as four separate and as yet unidentified genes may make a contribution to Late Onset Alzheimer's Disease (LOAD) that is as significant or more significant than that of APOE. To help researchers in their quest to identify these genes, the NIA plans to expand the National Cell Repository at the Indiana University Alzheimer's Disease Center.

The Repository was established in 1989 to provide genetic researchers with cell lines and/or DNA samples from people with well-documented family histories of AD. Many researchers working to identify genetic defects associated with AD have used genetic material supplied by participants in the Repository project.

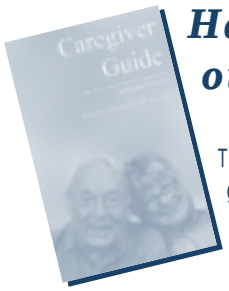
Identifying additional risk and prevention factor genes for LOAD will be important to understanding different etiologies, developing better models, and predicting risk of developing the disease in individuals of different genetic backgrounds. Efforts to identify risk factor genes are moving steadily forward using a variety of approaches. Three recent studies indicate that there may be as many as two novel LOAD gene loci on the long arm of chromosome 10. Finding new risk factor genes will help identify pathways affecting the development or progression of AD and may eventually lead to better predictors of the disease.

Studies on LOAD have shown that one form of the APOE gene on chromosome 19—APOE4—is a susceptibility factor for sporadic LOAD that modulates the risk of developing AD, rather than being its cause. To date, this is the only commonly accepted risk factor gene in the late-onset form of the disease.

Because most researchers rely on extended families, sibling pairs, or case-control studies to look for genes, the goal of expanding the repository will be to develop cell lines and collect data from persons identified through the Alzheimer's Disease Centers (ADCs) who fall within one of these three categories. This expanded catalogue of family and case control samples will assist in sample and data sharing. ADCs will be encouraged to bank cells and DNA from well-studied AD patients, at the repository.

In March 2002, the NIA's Neuroscience and Neuropsychology of Aging Branch will host a workshop on the genetics of LOAD, with a major focus being informing the genetics community about the new initiatives and encouraging sharing of data, phenotypic information, and biological resources. The workshop also will explore collaboration among grantees doing research on informed consent and other LOAD issues; and interaction with industry.

To contact the National Cell Repository at the Indiana University Alzheimer's Disease Center, call toll-free, 1-800-526-2839 or 1-317-274-2839, or e-mail: safox@iupui.edu, or visit http://medgen.iupui.edu/research/alzheimer/.



Have you ordered your free copy of our new Caregiver Guide?

The NIA's new *Caregiver Guide* is full of helpful tips for caregivers of people with AD. For your free copy, use the order form on the back of the newsletter to fax a request to ADEAR, or call us at 1-800-438-4380, M-F, 8:30 - 5:00 (ET).

Healthy Aging & Memory Study

(Continued from page 1)

Most will be self-administered by the volunteer and/or a study partner who has regular contact with him/her. The experimental instruments will measure cognition, activities of daily living, overall change, quality of life, and cost-effectiveness of treatments.

Another objective is to assess the accuracy of information gathered from subjects by mail, telephone, and the Internet, compared to that gathered in clinic visits. Participants will be randomized into two groups of 300 each to complete the new instruments either at home (with telephone assistance and returned by mail) or at their clinic visits.

An additional 50 participants who have access to a computer and the Internet will complete Web-based instruments. The aim of these data collection methods is to ease the burden of time and cost involved for volunteers and researchers in AD prevention trials. If these new methods and tools prove to be accurate and effective, they will help measure and track participants in AD clinical trials more efficiently and quickly.

Study sites are recruiting a total of 650 senior volunteers who are age 75 or older, in good health, with no memory problems, and English- or Spanish-speaking. Each volunteer must have a study partner (friend, family member, or other person) who has contact with him/her at least twice a week (in person or by phone), and can come with the volunteer on clinic visits over the course of the 4-year study.

Study participants will be actively recruited from minority populations, including: African American, Latino, Asian American, and Native American. People in these groups are urged to participate to ensure that the study findings are relevant for their communities. For more information, or to volunteer for the Healthy Aging & Memory Study, call the ADEAR Center at 1-800-438-4380, or visit www.alzheimers.org/trials.

Add-on AD Prevention Trial to Study Effects of Vitamin E and Selenium

Imagine the possibilities for AD research of having as many as 32,000 older men already enrolled in a long-term clinical trial studying the effects of vitamin E and selenium. A National Institute on Aging grantee did just that, when he saw the potential to create an AD clinical trial from within a National Cancer Institute (NCI) prostate cancer prevention trial already underway.

The NCI's SELECT Study, coordinated by the Southwest Oncology Group, is enrolling 32,000 participants to study the possibilities of preventing prostate cancer through use of vitamin E and selenium supplements. Vitamin E and selenium are antioxidants that protect cells against the effects of free radicals, which are produced during normal oxygen metabolism. Free radicals can damage cells and contribute to the development of some chronic diseases. Selenium also is essential for normal functioning of the immune system and thyroid gland. Plant foods are the major dietary sources of selenium in most countries throughout the world. Selenium also can be found in some meats and seafood.

Because observational studies have suggested that antioxidants also may help prevent AD, NIA has funded a study to recruit 10,000 men who are participating in the SELECT trial.

Some studies indicate that death from cancer, including lung, colorectal, and prostate cancers, is lower among people with higher selenium blood levels or intake, and incidence of nonmelanoma skin cancer is significantly higher in areas of the United States with low soil selenium levels.

Because observational studies have suggested that antioxidants also may help prevent AD, NIA has funded a study to recruit 10,000 men who are participating in the SELECT Trial, to enroll in the Prevention of Alzheimer's Disease with Vitamin E and Selenium (PREADVISE) Clinical Trial. Selenium and/or vitamin E may increase antioxidant defenses in the brain. The combination of these supplements has not been studied for brain cell protection or as prevention for AD. PREADVISE participants are asked to stay in the study for 7 to 12 years, and must be:

- enrolled in the SELECT study
- 62 years of age or older, or 60 if African-American or Hispanic
- in general good health with no neurological or psychiatric illness

A 5-minute memory check is performed once yearly. If the memory check suggests problems, a slightly longer examination will be offered. If that exam indicates problems, the participant may be asked to see a neurologist, geriatrician, or neuropsychiatrist. Participants visit their study site every 6 months to get new bottles of capsules. These capsules will contain selenium, vitamin E, both, or inactive capsules. Neither participants nor researchers will know which capsules participants take.

To participate in the NCI's Cancer Information Service at 1-800-4-CANCER (1-800-422-6237), or PREADVISE trial, contact the ADEAR Center at 1-800-438-4380. Information on both studies also is available at www.swog.org by clicking on the link titled SELECT. The ADEAR Center's Web site, www.alzheimers.org also has information about enrollment in the study.

Welcome Two New ADCs!

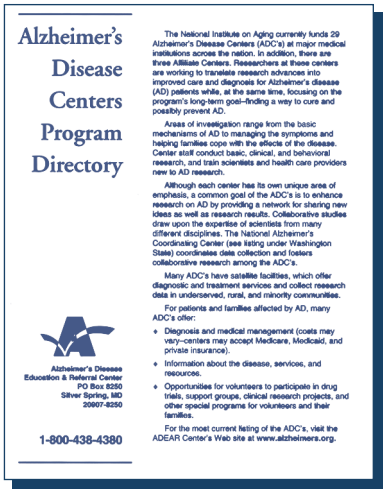
Two new NIA-funded Alzheimer's Disease Centers (ADCs) are now fully operational and providing research and services in Arizona and Arkansas. These new programs join 29 other ADCs across the country (plus three Affiliate Centers), providing a wide array of services to AD patients, family and professional caregivers, educators, researchers, and clinicians.

The Arizona ADC is located in Phoenix, directed by Eric Reiman, M.D., and affiliated with the Sun Health Research Institute/Arizona Consortium. The Arkansas ADC is located in Little Rock, directed by Cornelia M. Beck, R.N., Ph.D., and affiliated with the University of Arkansas for Medical Sciences.

New ADC Program Directory Available

The new, six-page ADC Program Directory lists all ADCs, State-by-State, and includes mail, telephone, e-mail, and Web site contact information for each. To order your free copy, please use the order form on the back page, or call the ADEAR Center at 1-800-438-4380.

The directory also is online and can be viewed at www.alzheimers.org/pubs/adcdir.html.



Alzheimer's Disease Centers Program Directory

The National Institute on Aging currently funds 29 Alzheimer's Disease Centers (ADC's) at major medical institutions across the nation. In addition, there are three Affiliate Centers. Researchers at these centers are working to translate research advances into improved care and diagnosis for Alzheimer's disease (AD) patients while, at the same time, focusing on the program's long-term goal–finding a way to cure and possibly prevent AD.

Areas of investigation range from the basic mechanisms of AD to managing the symptoms and helping families cope with the effects of the disease. Center staff conduct basic, clinical, and behavioral research, and train scientists and health care providers new to AD research.

Although each center has its own unique area of emphasis, a common goal of the ADC's is to enhance research on AD by providing a network for sharing new ideas as well as research results. Collaborative studies draw upon the expertise of scientists from many different disciplines. The National Alzheimer's Coordinating Center (see listing under Washington State) coordinates data collection and fosters collaborative research among the ADC's.

Many ADC's have satellite facilities, which offer diagnostic and treatment services and collect research data in underserved, rural, and minority communities.

For patients and families affected by AD, many ADC's offer:

- Diagnosis and medical management (costs may vary–centers may accept Medicare, Medicaid, and private insurance).
- Information about the disease, services, and resources.
- Opportunities for volunteers to participate in drug trials, support groups, clinical research projects, and other special programs for volunteers and their families.

For the most current listing of the ADC's, visit the ADEAR Center's Web site at www.alzheimers.org.

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CHID Highlights

CHID Highlights describes materials recently added to the Alzheimer's disease file of the Combined Health Information Database (CHID). The items selected represent topics and formats of general interest to readers of *Connections* and ADEAR Center users or their clients. Please order directly from the source listed for each item. Journal articles are available in many university and medical school libraries. CHID is accessible on the Internet at http://www.chid.nih.gov or by following the link at http://www.alzheimers.org, or by following the National Library of Medicine's link to CHID at http://www.nlm.nih.gov/medlineplus/databases.html.

Genetics Research

Decoding Darkness: The Search for the Genetic Causes of Alzheimer's Disease

Tanzi, R.E.; Parson, A.B. Perseus Publishing. 2000. 281 p.

Available from Perseus Books Group, Customer Service, 5500 Central Avenue, Boulder, CO 80301. 1-800-386-5656; FAX: 303-449-3356. Internet: http://www.perseuspublising.com. PRICE: $26.00.

This book presents a fascinating history of the medical journey to find the genetic causes of AD. It describes the experiences of neurogeneticist Rudolph Tanzi, an AD researcher, who in the early 1980s participated in a landmark experiment to identify AD genes. Delving into the laboratories of many prominent researchers, the book offers a view of the high stakes in molecular genetics and the families whose lives depend upon it. The authors conclude that AD may ultimately be effectively treated and even prevented.

Brain Diseases Guide

Brain Connections: Your Guide to Information on Brain Diseases and Disorders. Fifth Edition 2000-2002

Dana Alliance for Brain Initiatives. 2000. 49 p.

Available from the Dana Alliance for Brain Initiatives, 745 Fifth Avenue, Suite 700, New York, NY 10151. 212-223-4040; FAX: 212-317-8721. Internet: http://www.dana.org. PRICE: Free.

This resource guide lists organizations that assist people with brain diseases and disorders and those responsible for their care. It includes over 275 organizations, listed alphabetically by the disease or disorder. It emphasizes organizations that are national in scope. The guide offers telephone numbers, postal and e-mail addresses, web sites, and services available. The guide concludes with information about the Dana Press and ways that the reader can support brain research.

Professional Caregiving

Dementia and the Older Adult: The Role of the Geriatric Care Manager

Knutson, K., in: Cress, C. Handbook of Geriatric Care Management. Aspen Publishers, Inc. 2001. p. 207-254.

Available from Aspen Publishers, Inc., 7201 McKinney Circle, Frederick, MD 21704. 1-800-234-1660; FAX: 1-800-901-9075. Internet: http://www.aspenpublishers.com. PRICE: $49.00.

Business and clinical aspects of geriatric care management are discussed in this book. Chapter 12 focuses on the care of older adults with dementia. The chapter reviews the definition and diagnosis of dementia and explores the experience of dementia from the client's perspective. Then, it describes the role of the geriatric care manager, which includes interviewing the family and client, assessing the patient's functioning, developing a written plan of care, and managing the patient's ongoing care. The chapter concludes with suggested guidelines for communicating with other professionals and for training family and paid caregivers about dementia care. Appendices cover assessment instruments, how impairments impact abilities, and a referral checklist.

Communication in AD

Communication: How to Communicate With Someone Who Has Alzheimer's Disease or Related Dementia

Healing Arts Communication, Medford, OR. 2001. Videotape.

Available from Healing Arts Communication, 33 North Central, Suite 211, Medford, OR 97501. 1-888-846-7008; FAX: 541-858-6696. Internet: http://www.homecarecompanion.com. PRICE: $89.95.

This video reviews the role of communication in the care of a person with AD. The video defines AD and explains its impact on the brain and communication skills. The narrator stresses the importance of the person's feelings and states that the caregiver should avoid arguments or upsetting situations. The video then covers specific areas of communication, including vocabulary, repetitive questions, and aggressive behaviors. Other forms of communication that may work better than verbal skills include gestures, body language, tone of voice, and touch. The video then discusses the topics of how to answer difficult questions and how to visit when the person no longer recognizes loved ones. A list of helpful resources is provided.

April 3-6, 2002

7th International Geneva/Springfield Symposium on Advances in Alzheimer's Disease Therapy
Geneva, Switzerland

Contact:
South Illinois University School of Medicine
Office of CME
P.O. Box 19602
Springfield, IL 62794-9602
217-545-7342
http://www.siumed.edu/cme/geneva.pdf

April 4-7, 2002

Second Joint Conference of the National Council on the Aging and the American Society on Aging: Crossing the Great Divide - A Call for Compassion and Creativity
Denver, CO

Contact:
American Society on Aging
833 Market Street, Suite 511
San Francisco, CA 94103-1824
1-800-537-9728 or
415-974-9600
fax: 415-495-6509
info@asaging.org

April 6, 2002

Eighth Annual Update on Alzheimer's Disease and Other Dementias
Baltimore, MD

Contact:
Office of Continuing Medical Education
Johns Hopkins University School of Medicine
Turner 20, 720 Rutland Ave
Baltimore, MD 21205-2195
410-955-2959
Fax: 410-955-0807
cmenet@jhmi.edu

April 7-10, 2002

Vascular Factors in Alzheimer's Disease
Kyoto, Japan

Contact:
VFAD 2002 Secretariat
c/o JTB Communications Inc.
Sankei Building 7F
Umeda 2-4-9
Kitaku, Osaka 530-0001
Japan
81-6-6348-1391
fax: 81-6-6456-4105
vfad2002@jtbcomm.co.jp

April 8-10, 2002

AAHSA's Spring Conference & Exposition
Washington, DC

Contact:
American Association of Homes and Services for the Aging
2519 Connecticut Ave NW
Washington, DC 20008-1520
202-783-2242
202-783-2255
www.aahsa.org

April 13-20, 2002

2002 Annual Meeting of the American Academy of Neurology
Denver, CO

Contact:
American Academy of Neurology
1080 Montreal Avenue
St. Paul, MN 55116
651-695-1940
http://www.aan.com

April 15-18, 2002

14th Annual National Managed Health Care Congress: Managed Health Care - The Next Generation
Baltimore, MD

Contact:
National Managed Health Care Congress
71 Second Avenue, 2nd Floor
Waltham, MA 02451
781-663-6000
fax: 781-663-6414
www.nmhcc.org

April 19-20, 2002

ASPET - Ray Fuller Symposium: Alzheimer's Disease
New Orleans, LA

Contact:
American Society for Pharmacology and Experimental Therapeutics
9650 Rockville Pike
Bethesda, MD 20814-3995
301-530-7060
markin@aspet.org
http://www.aspet.org

April 21-27, 2002

7th Neurodegenerative Disorders: Common Molecular Mechanisms Conference
Montego Bay, Jamaica

Contact:
World Events Forum, Inc.
5030 N. Marine Drive, Suite 2608
Chicago, IL 60640
773-784-8134
fax: 208-575-5453
jamaica@worldeventsforum.com

April 27-30, 2002

14th Annual Alzheimer's Association Public Policy Forum
Washington, DC

Contact:
Alzheimer's Association
Public Policy Division
1319 F Street, NW, Suite 710
Washington, DC 20004-1106
202-393-7737
diane.blake@alz.org

May 2-3, 2002

Alzheimer's Disease: Update on Research, Treatment, and Care: UCSD Alzheimer's Disease Research Center's Annual Conference
San Diego, CA

Contact:
Alzheimer's Disease Research Center
UCSD
8950 Villa La Jolla Drive, Suite 1200
La Jolla, CA 92037
858-622-5800
fax: 858-622-1017
adrc@ucsd.edu

May 8-12, 2002

2002 Annual Scientific Meeting: Shaping the Future for Older Adults
Washington, DC

Contact:
American Geriatrics Society
The Empire State Building
350 Fifth Avenue, Suite 801
New York, NY 10118
212-308-1414

May 10-12, 2002

Statistical Methodology in Alzheimer's Disease Research II
Lexington, KY

Contact:
Alzheimer's Disease Research Center
Sanders-Brown Center on Aging
101 Sanders-Brown Building
University of Kentucky
Lexington, KY 40536-0230
859-257-1412
fax: 859-323-2866

May 14, 2002

Schreiber Memorial Conference for Alzheimer's Caregivers
Kalamazoo, MI

Contact:
Alzheimer's Association - Michigan Great Lakes Chapter
3810 Packard Road, Suite 240
Ann Arbor, MI 48108
734-677-3081 or 1-800-337-3827
fax: 734-677-3091

May 25-30, 2002

AAPA's 30th Annual Physician Assistant Conference
Boston, MA

Contact:
American Academy of Physician Assistants
950 North Washington Street
Alexandria, VA 22314-1552
703-836-2272
fax: 703-684-1924
www.aapa.org

June 17-20, 2002

Summer Series on Aging - West Coast
San Francisco, CA

Contact:
American Society on Aging
833 Market Street, Suite 511
San Francisco, CA 94103-1824
1-800-537-9728
www.asaging.org/summer-series

June 20-23, 2002

Annual Meeting of the American Association of Neuropathologists
Denver, CO

Contact:
American Association of Neuropathologists
Department of Laboratory Medicine and Pathology
Mayo Clinic, 200 First St., SW
Rochester, MN 55905
507-284-3394
fax: 507-284-1599
aanp@mayo.edu

July 13-16, 2002

7th European Congress of Neuropathology
Helsinki, Finland

Contact:
Congress Secretariat
CONGREX / Blue & White Conferences Oy
PO Box 81
Sulkapolku 3 (3rd floor)
FIN-00371
Helsinki, Finland
+358-9-560 7500
fax: +358-9-560 75020
neuropathology2002@congrex.fi
www.congrex.fi/neuropatholgy 2002/

July 20-25, 2002

8th International Conference on Alzheimer's Disease and Related Disorders
Stockholm, Sweden

Contact:
Alzheimer's Association
International Research Conference
PO Box A3498
Chicago, IL 60690-9564
312-335-5813
fax: 312-335-5781
www.alz.org/internationalconference

October 12-14, 2002

Third International Conference on Family Care: Empowerment Through Innovation.
Washington, DC

Contact:
National Alliance for Caregiving
4720 Montgomery Lane, Suite 642
Bethesda, MD 20814
301-718-8444
fax: 301-652-7711
www.caregiving.org

Order Form

Alzheimer's Disease Education and Referral Center

❑ *AD Fact Sheet* (No. Z-12) **updated*

❑ *Caregiver Guide* (No. Z-169) **new*

❑ *ADC Program Directory* (No. Z-01) **updated*

You may ask the ADEAR Center for a topical search (a list of materials) on another subject related to Alzheimer's disease. Outline the subject for your search in the space below.

_____

_____

_____

To ensure that we can contact you with any questions regarding the search, please provide a daytime telephone number: (_____)_____.

Name: _____

Title: _____

Organization: _____

Address: _____

_____

❑ Check here if you are a health professional or a professional caregiver and would like your name added to the ADEAR Center mailing list to receive future issues of *Connections*.

To order any of the above materials, send this page to:

ADEAR Center, PO Box 8250, Silver Spring, MD 20907-8250

You also may call our toll-free telephone number: 1-800-438-4380; reach us by fax: 301-495-3334; or contact us on the Internet: e-mail: adear@alzheimers.org, and Web site: http://www. alzheimers.org

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NATIONAL INSTITUTES OF HEALTH
BUILDING 31, ROOM 5C27
31 CENTER DR MSC 2292
BETHESDA, MD 20892-2292

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