

The National Cell

Repository is a repository for families with Alzheimer's Disease or severe memory loss. Families having two or more living individuals with memory loss are encouraged to participate. We would like to thank the hundreds of families nationwide who are already participating in the National Cell Repository. Many family members have provided blood samples, which researchers use to study Alzheimer's disease (AD) and other related diseases. Our hope is that, through the efforts of our participants, we will one day unravel the mystery of devastating diseases, like AD. We are always eager to accept new families to help us move toward this goal.

National Cell Repository for Alzheimer's Disease

Department of Medical Genetics

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NCRAD Update

Newsletter of the National Cell Repository for Alzheimer's Disease Volume 6 ■ March 2005



National Cell Repository for Alzheimer's Disease – Fifteen Years of Research

By Tatiana Foroud, Ph.D.

Indiana University School of Medicine

The National Cell Repository for Alzheimer's Disease (NCRAD) is celebrating its 15th anniversary in 2005. With the participation of the more than 870 families in NCRAD we have significantly advanced Alzheimer's disease research. In the past 15 years, NCRAD has sent approximately 10,000 biological samples to Alzheimer's disease researchers. Importantly, data and samples from our families have resulted in 82 different scientific publications.

While in this column we can not describe all of the research that has been performed with samples from NCRAD, we'd like to tell you some of the highlights. First, families working with NCRAD were critical in helping researchers understand the role of the apolipoprotein E (APOE) gene in Alzheimer's disease. As we have discussed in previous newsletters (Volume 1, February 2002, Volume 2, October 2002 and Volume 4, January 2004; to download previous newsletters go to the NCRAD website at www.ncrad.org) there are 3 different forms of the APOE gene that differ in their DNA sequence. Inheriting one particular form of this gene, termed APOE4, increases the risk of developing Alzheimer's disease, while inheriting a different form of this gene termed APOE2 decreases the risk of developing Alzheimer's disease. Studies using data from families in NCRAD have helped researchers determine that while APOE is an important genetic risk factor for Alzheimer's disease, there are clearly other genes that are also important in determining the risk for developing Alzheimer's disease.

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Fifteen Years of Research

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Additional studies by a number of researchers have targeted several chromosomal regions that appear likely to include a gene or genes that might be important in determining susceptibility to Alzheimer's disease. These important chromosomes include chromosomes 9, 10 and 12. Researchers working with data from families participating in NCRAD have been actively studying a number of different genes on these chromosomes to determine which are important in Alzheimer's disease. While no additional genes have been conclusively shown to be important in Alzheimer's disease susceptibility, there are many ongoing studies that we hope will lead to new research results.

Another important area of research has been studying what factors are important in determining when an individual develops Alzheimer's disease. There is great variability in the onset of Alzheimer's disease symptoms. Even among families with what is called "late onset" Alzheimer's disease, in which symptoms develop after age 60, there are some individuals who develop Alzheimer's disease at age 60 while others only develop symptoms in their 70s or 80s.

A number of researchers have performed studies using data and families from NCRAD to begin to identify genes that influence when an individual develops Alzheimer's disease. No definitive genes have yet been reported, but there are several important leads.

Finally, another area in which NCRAD families have been very important has been the study of early onset Alzheimer's disease and familial dementia that is not Alzheimer's disease. Individuals who develop Alzheimer's disease before the age of 60 are described as having early onset Alzheimer's disease. In some families, the disease is caused by a DNA sequence change in one of three genes termed presenilin I (PS1), presenilin II (PS2) or amyloid precursor protein (APP) (previously discussed in newsletters Volume 1, February 2002, Volume 2, October 2002 and Volume 4, January 2004). While mutations in these genes cause Alzheimer's disease in fewer than 5% of individuals with Alzheimer's disease, their discovery has led researchers to a greater understanding of the changes in the brain that result in Alzheimer's disease. Another important area of research has been the study of



Tatiana Foroud, PH.D.

families with dementia that is not caused by Alzheimer's disease. The NCRAD study includes families with these often rare familial dementias and they have been essential to our greater understanding of several genes which can cause symptoms similar to Alzheimer's disease.

The participation of families with Alzheimer's disease is critically important for scientific research which seeks to understand why individuals develop Alzheimer's disease in the hopes that this understanding will lead to preventions and cures for the disease. The researchers using data and samples from NCRAD families are extremely grateful for the participation of our families. Thank you!

*Check Out Our
New Website!*

www.ncrad.org

The new NCRAD website, www.ncrad.org, is an online resource for anyone wanting to learn more about the National Cell Repository for Alzheimer's Disease. This site has information for families wanting to get involved with NCRAD and the new NIA Alzheimer's Disease Genetics Study, links to related sites, and contacts for NCRAD staff.

Part of the website is for researchers and coordinators who are involved in the NIA Alzheimer's Disease Genetics Study or who are interested in obtaining samples from NCRAD. The researcher and coordinator section of the website is password protected and only available to qualified researchers.

Alzheimer's Disease Medications

Fact Sheet

-Reprinted with permission from the Alzheimer's Disease Education and Referral (ADEAR) Center

Five prescription drugs currently are approved by the U.S. Food and Drug Administration to treat people who have been diagnosed with Alzheimer's disease (AD). Treating the symptoms of AD can provide patients with comfort, dignity, and independence for a longer period of time and can encourage and assist their caregivers as well. It is important to understand that none of these medications stops the disease itself.

Treatment for Mild to Moderate AD

Four of these medications are called cholinesterase inhibitors. These drugs are prescribed for the treatment of mild to moderate AD. They may help delay or prevent symptoms from becoming worse for a limited time and may help control some behavioral symptoms. The medications are: Reminyl® (galantamine), Exelon® (rivastigmine), Aricept® (donepezil), and Cognex® (tacrine). Scientists do not yet fully understand how cholinesterase inhibitors work to treat AD, but current research indicates that they prevent the breakdown of acetylcholine, a brain chemical believed to be important for memory and thinking. As AD progresses, the brain produces less and less acetylcholine; therefore, cholinesterase inhibitors may eventually lose their effect.

No published study directly compares these drugs. Because all four work in a similar way, it is not expected that switching from one of these drugs to another will produce significantly different results. However, an AD patient may respond better to one drug than another. Cognex® (tacrine) is no longer actively marketed by the manufacturer.

Treatment for Moderate to Severe AD

The fifth approved medication, known as Namenda® (memantine), is an N-methyl D-aspartate (NMDA) antagonist. It is prescribed for the treatment of moderate to severe AD. Studies have shown that the main effect of Namenda® is to delay progression of some of the symptoms of moderate to severe AD. The medication may allow patients to maintain certain daily functions a little longer. For example, Namenda® may help a patient in the later stages of AD maintain his or her ability to go to the bathroom independently for several more months, a benefit for both patients and caregivers. Namenda® is believed to work by regulating glutamate, another important brain chemical that, when produced in excessive amounts, may lead to brain cell death. Because NMDA antagonists work very differently from cholinesterase inhibitors, the two types of drugs can be prescribed in combination.



“ It is important to follow the doctor's instructions when taking any medication, including vitamins and herbal supplements. Also, let the doctor know before adding or changing any medications. ”

Dosage and Side Effects

Doctors usually start patients at low drug doses and gradually increase the dosage based on how well a patient tolerates the drug. There is some evidence that certain patients may benefit from higher doses of the cholinesterase inhibitor medications. However, the higher the dose, the more likely are side effects. The recommended effective dosage of Namenda® is 20 mg/day after the patient has successfully tolerated lower doses.

Patients may be drug sensitive in other ways, and they should be monitored when a drug is started. Report any unusual symptoms to the prescribing doctor right away. It is important to follow the doctor's instructions when taking any medication, including vitamins and herbal supplements. Also, let the doctor know before adding or changing any medications.

Dementia with Lewy Bodies: Not Exactly Alzheimer's or Parkinson's Disease

by Daniel I. Kaufer, MD*

Alzheimer's disease (AD) is the most common cause of middle and late-life dementia, accounting for 50-60% of all cases. Until recently, vascular dementia (formerly known as multi-infarct dementia) was thought to be the second most common type of dementia, either alone or in combination with AD. However, over the last decade and a half there has been increasing recognition of another disorder which is now thought to be the second most frequent cause of dementia. In various autopsy studies performed throughout the world, this form of dementia, referred to as "Dementia with Lewy Bodies" (DLB), has been observed to represent 15-25% of all cases. The fact that we now recognize DLB as a distinct entity highlights the important information that can only be learned through post-mortem (autopsy) examinations.

As with AD, there is no non-invasive diagnostic test for DLB. To make the diagnosis with certainty, one has to examine different areas of the brain (usually after death) for Lewy bodies, which are small, round inclusions that are found within nerve cells. It has been known for over 70 years that Lewy bodies are a characteristic feature of another degenerative brain disorder, Parkinson's disease (PD).

Although PD and DLB both involve the accumulation of Lewy bodies, in DLB this occurs in both the brainstem and cortical areas of the cerebral hemispheres, whereas only the brainstem is significantly affected in PD.

In addition to its pathological similarities to PD, DLB also is commonly associated with pathological changes that characterize AD. Of the two main pathological hallmarks of AD, amyloid plaques and neurofibrillary tangles, plaques also frequently occur in patients with DLB, whereas tangles are less commonly seen. As such, DLB exhibits pathological features that can overlap with both AD and PD. This fact led to vigorous debate over the last decade about what to call the disorder—some viewed it primarily as a form of AD, whereas others viewed it as a form of PD, and still others viewed it as a unique entity. Although not completely resolved, a consortium of international researchers met and recently suggested guidelines for diagnosing the disorder, which they agreed to call "Dementia with Lewy bodies."

Just as DLB has pathological features in common with both AD and PD, it may also resemble these two disorders clinically. On the one hand, short-term memory deficits, visuospatial difficulties, and language disturbances (as seen in AD) may be present, as may some of the classic manifestations of PD—resting tremor, motor slowing, rigidity, reduced facial expression, stooped posture, and shuffling gait. However, DLB has some distinguishing clinical features of its own.

Diagnosis Criteria

As proposed in the guidelines published by the Consortium on DLB, there are three types of criteria for diagnosing DLB:

- First, a mandatory requirement is that a significant degree of cognitive impairment in two or more areas (i.e., a dementia) must be present.
- Second, two or more of the following core features must be present: 1) marked and persistent fluctuations in attention or level of alertness, 2) well-formed visual hallucinations, and 3) extrapyramidal motor signs (as may be seen in PD).
- Third, various supportive features may also suggest the diagnosis, including repeated falls, unexplained syncope or brief periods of loss of consciousness, other psychotic features such as delusions, sleep disturbances (e.g. sleeping excessively, abnormal movements during sleep), and sensitivity to neuroleptics, a class of medication used to treat psychotic symptoms.

The last feature may be particularly problematic, as psychotic symptoms can be severe, and individuals with DLB often are very sensitive to the extrapyramidal motor side effects associated with many neuroleptics. Newer antipsychotic drugs, such as risperidone (Risperdal) and olanzapine (Zyprexa), may be better tolerated by individuals with DLB. In our experience, psychotic symptoms, particularly visual hallucinations, may also benefit from cholinesterase-inhibitor agents such as tacrine (Cognex) and donepezil (Aricept). If extrapyramidal motor signs are prominent in an individual with DLB, moderate use of anti-Parkinson's drugs to enhance dopamine function can be helpful, but may exacerbate psychotic symptoms.

Beyond the desire to minimize or avoid exposing DLB patients to neuroleptic drugs, another important reason for distinguishing DLB from AD or PD patients is that they may particularly benefit from cholinesterase-inhibitor therapy. Consistent with our experience, several reports have documented frequently good, and occasionally dramatic responses of DLB patients to tacrine or donepezil treatment. This may be explained by the observations that DLB typically involves a greater loss of the brain chemical acetylcholine compared to AD, but is associated with fewer neurofibrillary tangles (which destroy nerve cells). Through ongoing clinical, pathological, and therapeutic research investigations, it is hoped that an increased understanding of DLB and its relationship to both AD and PD will lead to more specific and effective interventions for all three disorders.

**Dear Friends* (newsletter), Summer 1997, Alzheimer Disease Research Center, University of Pittsburgh. Reprinted here with the permission of the Alzheimer Disease Research Center of the University of Pittsburgh

Dementia Diagnosis

-Courtesy of the Washington University Alzheimer's Disease Research Center

Dementia is the general loss of intellectual abilities including impaired memory, judgment, and abstract thinking in an otherwise alert person. Many illnesses, some progressive in nature, that adversely affect the brain can cause dementia. In late life, however, by far the most common cause of dementia is Alzheimer's disease (AD). A definite diagnosis of Alzheimer's disease still requires the microscopic examination of brain tissue, generally obtained at autopsy. Nonetheless, much has been learned about the symptoms and signs of AD and with the appropriate clinical methods physicians often can make an accurate diagnosis. The initial step is to assess whether dementia indeed is present. If it is, then a differential diagnosis determines whether it is due to AD or another dementing disorder. The clinical diagnostic approach includes a careful history of the illness, physical and neurological examinations, mental status testing, and specific laboratory procedures, including neuroimaging studies.

HISTORY

INTERVIEW

To determine whether the individual is demented, someone who knows the patient well (eg, the spouse or an adult child) is interviewed to learn how the individual's memory, thinking, and behavior, have changed from prior performance. That is, is the individual impaired in his or her ability to carry accustomed activities (e.g., job; driving; managing finances; cooking; maintaining hobbies; communication; social behavior) because of memory and thinking problems? Did the changes noted by the informant come on slowly or abruptly? Have they steadily worsened or have there been fluctuations in behavior?

Significant changes in memory and thinking that interfere with everyday activities (difficulty driving, functioning at work, interactions with family and peers, etc.) signal a disease state, not usual aging. Changes due to usual aging relate to limited attentional resources ("I forgot what I came in here to get") or to diminished speed of information processing ("I couldn't remember his name until later"). Such changes usually do not worsen over time nor do they interfere with everyday activities.

The presence of physical decline (vision or hearing loss) should be explored to be sure it is not responsible for the problem. During the history portion of the exam, risk factors should be noted and other medical problems that might complicate the diagnosis.



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MEDICATIONS

The patient's medication history should be reviewed for drugs that may interfere with memory and thinking. These would include certain pain, sleep, psychiatric or urination medications. In addition, non-medication drugs such as alcohol or illicit substances may interfere with memory.

PHYSICAL EXAMINATION

Although the physical examination for a dementia patient is usually normal, the examination can sometimes yield clues about the cause of dementia or other conditions that may exacerbate dementia (e.g., profound hearing loss isolates the patient). In more advanced stages of dementia, a neurologic examination may reveal motor and reflex dysfunction.

TESTING

OFFICE

Paper and pencil tests of memory can provide the physician with more specific information regarding the patient's memory and thinking. Brief instruments are intended to measure cognitive function objectively, but can not

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Dementia Diagnosis

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be used alone as diagnostic tools. Often mildly demented, highly educated individuals score in the normal range on these tests, whereas poorly educated, non-demented persons may score in the impaired range. More extensive memory and thinking tests can be used to determine how severe (what stage) the dementia is or identify specific brain regions which are not functioning normally.

LABORATORY

Laboratory tests can identify conditions that potentially contribute to cognitive impairment, some that may be reversible. Some lab testing may include: vitamin B12 urinalysis, chest xray, electrocardiography, blood cell count, blood chemistry, and measurement of thyroid stimulating hormone. Depending on what the physician finds during the history and physical examination, selected other studies may be indicated.

BRAIN IMAGING

Brain imaging technology is improving rapidly but standard methodologies cannot diagnose Alzheimer's disease early in its course. However such technologies can exclude other conditions such as normal pressure hydrocephalus, tumors or vascular complications (infarct). Common brain imaging methods include Computed Tomography (CT), Magnetic Resonance Imaging (MRI), Positron Emission Tomography (PET), and Single Photon Emission Computed Tomography (SPECT).

DIFFERENTIAL DIAGNOSIS

As stated earlier, there are many causes of dementia. Complicating the diagnosis is the possibility of mixed dementia when two causes may be present at the same time. Only 50-60% of clinically diagnosed cases of Alzheimer's disease are "pure" without other complicating conditions. Strokes and Lewy body disease (related to Parkinson's disease) each occur in about 25% of Alzheimer's disease cases.

Additionally, there are treatable conditions that mimic the symptoms and signs of dementia, including depression, delirium, and medication effects, etc. In arriving at a diagnosis, the physician will use all available information to make a differential diagnosis regarding the cause of the dementia.

Alzheimer's Books We Recommend:

Voices Of Alzheimer's: Courage, Humor, Hope, and Love in the Face of Dementia

by Betsy Peterson

- Paperback: 255 pages
- Publisher: Da Capo Press (November 1, 2004)
- ISBN: 0738209627

Aging with Grace

By David Snowdon, PhD

- Paperback: 256 pages
- Publisher: Bantam (April 30, 2002)
- ISBN: 0553380923

Living with Grief: Alzheimer's Disease

by Kenneth J. Doka (Editor)

- Paperback: 290 pages
- Publisher: Hospice Foundation of America (April, 2004)
- ISBN: 1893349055

The Alzheimer's Health Care Handbook: How to Get the Best Medical Care for Your Relative with Alzheimer's Disease, in and out of the Hospital

by Mary S. Mittelman, Cynthia Epstein

- Paperback: 208 pages
- Publisher: Marlowe & Company (September 1, 2003)
- ISBN: 1569244456

The 36-Hour Day

by Nancy L. Mace, MA, Peter V. Rabins, MD, MPH

- Paperback: Hardcover: 339 pages
- Publisher: Johns Hopkins University Press; 3rd edition (April 1, 1999)
- ISBN: 080186148

Free Caregiver Guide Available

The National Cell Repository for AD has over 250 Caregiver Guides available for interested families. This booklet was published by the National Institute on Aging and it is a great resource for a caregiver of someone with Alzheimer's disease. Topics covered include communication, exercise, sleep problems, wandering, visiting a person with AD, and choosing a nursing home, to name only a few.

Those interested in receiving a Caregiver Guide may call us at 1-800-526-2839. Get one while supplies last!

Research Opportunities

Cholesterol Lowering Agent to Slow Progression (CLASP) of Alzheimer's Disease Study

- Purpose: To investigate the safety and effectiveness of simvastatin (a cholesterol lowering drug or statin) to slow the progression of AD.
- Eligibility: Ages 50+ with mild to moderate AD
- Locations: AL, AZ, CA, CT, DC, FL, GA, IL, IN, KY, MA, MI, MN, MS, MO, NY, NC, OR, PA, RI, SC, TX, VT, WA
- Contact: NCRAD staff
PH: 800-526-2839 (toll free)
E-mail: alzstudy@iupui.edu

VITAL – VITamins to Slow Alzheimer's Disease (Homocysteine study)

- Purpose: To determine whether reduction of homocysteine levels with high-dose folic acid, B6, and B12 supplementation will slow the rate of cognitive decline in persons with AD.
- Eligibility: Ages 55+ with probable AD
- Locations: AL, AZ, CA, CT, DC, FL, GA, IL, IN, MA, MI, NV, NJ, NY, OH, OR, PA, RI, SC, TX
- Contact: NCRAD staff
PH: 800-526-2839 (toll free)
E-mail: alzstudy@iupui.edu

Alzhemed™ in Patients with Mild to Moderate Alzheimer's Disease

- The purpose of this Phase III study is to evaluate the efficacy and safety of Alzhemed™ compared to placebo (inactive substance pill) in patients with mild to moderate Alzheimer's disease.
- Eligibility: Age 50 +, both genders, with a diagnosis of Probable AD, treated with conventional AD therapies and must be on stable dose for at least four months prior to the screening visit and during the entire study period
- Locations: AZ, AR, CA, CO, CT, DC, FL, GA, IL, MA, MI, NJ, NY, NC, OH, OK, PA, RI, SC, TN, TX, VT, Alberta, British Columbia, New Brunswick, Nova Scotia, Ontario, Quebec
- Contact: E-mail only:
pic@neurochem.com

Huperzine A in Alzheimer's Disease

- Purpose: To evaluate the safety and efficacy of the Chinese herb huperzine A in the treatment of Alzheimer's disease (AD) in a randomized controlled trial of its effect on cognitive function.
- Eligibility: Age 55 + with probable AD, stable condition 3 months prior to screening. If interested, speak with contact about other eligibility requirements.
- Locations: AL, CA, DC, FL, GA, IL, NV, NJ, NY, NC, OR, PA, SC, TX
- Contact: Carolyn Ward, MSPH
PH: 202-784-6671
E-mail: cw2@georgetown.edu

Treatment of Agitation/Psychosis in Dementia/Parkinsonism (TAP/DAP)

- Purpose: To determine the efficacy (as well as safety, tolerability, and influence on parkinsonism) of quetiapine and donepezil, used alone or in combination, for the treatment of psychosis and/or agitation in patients with primary dementia complicated by coexistent parkinsonism.
- Eligibility: 50+, both genders, diagnosis of AD, symptoms of psychosis, agitation, parkinsonism
- Locations: AL, AZ, CA, GA, IL, MD, MA, MI, NV, NY, PA, TX, VT, WA
- Contact: Kimberly Schafer, M.S.
PH: (858) 622-5863
E-mail: kschafer@ucsd.edu

ALADDIN (VP 104): Antigonadotropin-Leuprolide in Alzheimer's Disease Drug Investigation Study

- Purpose: to investigate the safety and effectiveness of leuprolide (a hormone drug) to improve the cognitive function and slow the progression of Alzheimer's disease (AD)
- Eligibility: Age 65+, men, with mild to moderate Alzheimer's disease
- Locations: CA, CT, FL, MA, SC, VA, WI

- Contact: NCRAD staff
PH: 1-800-526-2839
E-mail: alzstudy@iupui.edu

Prevention of Alzheimer's Disease by Vitamin E and Selenium (PREADVISE)

- Purpose: As a prevention trial, PREADVISE is trying to find out if taking selenium and/or Vitamin E supplements can help to prevent memory loss and dementia such as Alzheimer's disease.
- Eligibility: Ages: 60 - 90, Male. Accepts Healthy Volunteers
- Locations: AL, AK, CA, CO, DC, FL, GA, IA, KS, KY, MD, MA, MI, MN, MS, MO, MT, NE, NV, NJ, NY, OH, OK, PA, SD, TN, TX, WA, WI, CANADA, PUERTO RICO
- Contact: Cecil R. Runyons
PH: 1-859-257-1412 Ext. 235
E-mail: preadvise@lsv.uky.edu

Valproate in Dementia (VALID)

- Purpose: To demonstrate whether valproate therapy delays the emergence of agitation and/or psychosis in outpatients with probable Alzheimer's disease (AD) who have not experienced agitation and psychosis in their illness. A secondary aim is to determine whether valproate therapy delays the progression of cognitive and functional measures of illness. This trial will also assess the tolerability and safety of low-dose, long-term valproate therapy.
- Eligibility: Ages 55 - 90 with probable AD
- Locations: CA, CT, DC, FL, GA, IL, MI, MO, NV, NY, OH, PA, RI, SC, TN, TX, VT, VA
- Contact: Laura Jakimovich, RN, MS
PH: 585-760-6578
E-mail:
laura_jakimovich@urmc.rochester.edu

Sources for Information and Support

Alzheimer's Association

<http://www.alz.org>

Tel: 312-335-8700 800-272-3900

Alzheimer's Disease Education and Referral Center (ADEAR)

<http://www.alzheimers.org>

Tel: 301-495-3311 800-438-4380

‡ ADEAR lists all 29 Alzheimer Disease Centers (ADCs) and their contact information.

Depression and Related Affective Disorders Association

<http://www.med.jhu.edu/drada>

Tel: 410-955-4647

Family Caregiver Alliance

<http://www.caregiver.org>

Tel: 415-434-3388 800-445-8106

National Parkinson Foundation

<http://www.parkinson.org/>

Tel: 305-547-6666 800-327-4545

Parkinson's Disease Foundation

(PDF)

<http://www.parkinsons-foundation.org>

Tel: 212-923-4700, 800-457-6676

Society for Progressive Supranuclear Palsy

<http://www.psp.org>

Tel: 410-486-3330 800-457-4777

National Organization for Rare Disorders (NORD)

<http://www.rarediseases.org>

Tel: 203-746-6518 800-999-NORD (6673)

Centers for Disease Control and Prevention (CDCP)

<http://www.cdc.gov>

Tel: 800-311-3435

Creutzfeldt-Jakob (CJD) Foundation Inc.

<http://cjdfoundation.org>

Tel: 954-704-0519 305-891-7579

10 Signs of AD

1. Memory loss.
2. Difficulty performing familiar tasks.
3. Problems with language.
4. Disorientation to time and place.
5. Poor or decreased judgment.
6. Problems with abstract thinking.
7. Misplacing things.
8. Changes in mood or behavior.
9. Changes in personality.
10. Loss of initiative.

If you recognize several of these warning signs in yourself or a loved one, the Alzheimer's Association recommends consulting a physician. Early diagnosis of Alzheimer's disease or other disorders causing dementia is an important step in getting appropriate treatment, care, and support services.

For more information, call the Alzheimer's Association at (800) 272-3900.



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