#### Alzheimer's Disease

#### **Goals & Objectives**

#### **Course Description**

"Alzheimer's Disease" is an asynchronous online continuing education course for physical therapists and physical therapist assistants. This course presents updated information about Alzheimer's Disease including sections on pathophysiology, etiology, symptomology, diagnosis, treatment, caregiving, and safety.

#### **Course Rationale**

The purpose of this course is to present current information about Alzheimer's Disease. Physical therapy professionals will find this information pertinent and useful when creating and implementing rehabilitation programs and home safety programs that address the challenges and needs specific to individuals with AD.

#### **Course Goals and Objectives**

Upon completion of this course, the participant will be able to

- 1. recognize the societal and economic impact of AD
- 2. identify the neuro-physiologic changes associated with AD
- 3. identify the current theories relating to AD etiology
- 4. name the risk factors for AD
- 5. list the symptoms of AD including its effects upon cognitive abilities required for safe and effective rehabilitative care.
- 6. identify current mechanisms utilized to diagnose AD
- 7. differentiate all of the current options available for treating AD
- 8. describe the responsibilities and challenges associated with being a caregiver
- 9. define a safe home environment for individuals with AD
- 10. identify the resources available for AD patients and their families

Course Provider - Innovative Educational Services

Course Instructor - Michael Niss, DPT

Target Audience – Physical therapists and physical therapist assistants

Course Educational Level - Introductory / intermediate

Course Prerequisites - None

**Method of Instruction/Availability – Online text-based course available continuously.** 

Criteria for Issuance of CE Credits - A score of 70% or greater on the course post-test.

Continuing Education Credits - 4 hours

**Determination of Credits** - Mergener Formula:  $.9 \times [-22.3 + (0.00209 \times 89,715) + (2.78 \times 20) + (15.5 \times 3)] = 240 \text{ minutes} = 4.0 \text{ hours}$ 

Fees - \$39.95

**Conflict of Interest** – No conflict of interest exists for the presenter or provider of this course.

**Refund Policy** - Unrestricted 100% refund upon request. The request for a refund by the learner shall be honored in full without penalty or other consideration of any kind. The request for a refund may be made by the learner at any time without limitations before, during, or after course participation.

# Alzheimer's Disease

# **Course Outline**

	Page(s)	
Goals and Objectives Outline Overview Prognosis Epidemiology Cost Pathophysiology Etiology Genetics Lifestyle Cholesterol and Homocysteine Tau Oxidative Damage Inflammation Aluminum Copper and Zinc	1 2 3 3 3-4 4 4-5 6-10 6-8 8 8 8-9 9 9	start hour 1
Symptomology Stages of AD Diagnosis New Guidelines Diagnostic Tools Treatment Medications	10-12 12-15 15-20 15-17 17-20 20-24 20-23	end hour 1 start hour 2
Other Approaches Caregiving Caring for a Person with AD Communication Activities of Daily Living Exercise Tailored Activity Program Safety Recommendations Supervision	23-24 24-30 25-26 27 27-30 30 31-44 31	end hour 2 start hour 3
Home Safety Behavior Management Sensory Impairment Driving Natural Disaster Safety Stigma of Alzheimer's Disease Research on Prevention Physical Activity Dietary Factors Social & Cognitive Activities Supplemental Information Resources References	32-36 36-40 40-42 42-43 43-44 45 45-48 45-46 46-47 47-48 49 50	end hour 3 start hour 4
Post-Test	52-53	end hour 4

### **Overview**

Alzheimer's disease is a devastating disorder of the brain's nerve cells that impairs memory, thinking and behavior and leads, ultimately, to death. The impact of Alzheimer's on individuals, families and our health care system makes the disease one of our nation's greatest medical, social and economic challenges.

Alzheimer's disease (AD) is the most common form of dementia among older people. It involves the parts of the brain that control thought, memory, and language.

AD is named after Dr. Alois Alzheimer, a German doctor. In 1906, Dr. Alzheimer noticed changes in the brain tissue of a woman who had died of an unusual mental illness. He found abnormal clumps (now called amyloid plaques) and tangled bundles of fibers (now called neurofibrillary tangles). Today, these plaques and tangles in the brain are considered hallmarks of AD.

Scientists also have found other brain changes in people with AD. There is a loss of nerve cells in areas of the brain that are vital to memory and other mental abilities. There also are lower levels of chemicals in the brain that carry complex messages back and forth between nerve cells. AD may disrupt normal thinking and memory by blocking these messages between nerve cells.

#### **Prognosis**

Alzheimer's disease is a slow disease, starting with mild memory problems and ending with severe brain damage. The course the disease takes and how fast changes occur vary from person to person. On average, AD patients live from 8 to 10 years after they are diagnosed, though the disease can last for as many as 20 years.

# **Epidemiology**

Alzheimer's disease presents a major health problem for the United States because of its enormous impact on individuals, families, the health care system, and society as a whole.

According to recent estimates, as many as 2.4 million to 5.1 million Americans have Alzheimer's disease. Unless the disease can be effectively treated or prevented, the number of people with AD will increase significantly if current population trends continue. That's because the risk of AD increases with age, and the U.S. population is aging. The number of people age 65 and older is expected to grow from 39 million in 2008 to 72 million in 2030, and the number of

people with AD doubles for every 5-year interval beyond age 65.

These numbers are significant now and will become even more so in the future because of dramatic increases in life expectancies since the turn of the century. Furthermore, the group over 85 - the group with the highest risk of AD - is the fastest growing group in the population.

#### Cost

The increasing number of people with AD and the costs associated with the disease mean that AD puts a heavy economic burden on society.

The total annual costs to all payers for the care of people with Alzheimer's disease will increase from \$172 billion in 2010 to \$1.08 trillion in 2050. Medicare costs for the care of people with Alzheimer's will increase more than 600 percent, from \$88 billion in 2010 to \$627 billion in 2050. Medicaid costs will increase 400 percent, from \$34 billion in 2010 to \$178 billion in 2050. Out-of-pocket costs to people with Alzheimer's and their families will increase more than 400 percent, from \$30 billion in 2010 to \$157 billion in 2050.

The cost of care is not only financial. Families, friends, and caregivers struggle with great emotional and physical stress as they cope with the physical and mental changes in their loved ones. Caregivers must juggle many responsibilities and adjust to new and changing roles. As the disease gets worse and caring at home becomes increasingly difficult, family members face difficult decisions about long-term care. The number of caregivers - and their needs - will steadily grow as our population ages and the number of people with AD increases.

# **Pathophysiology**

In healthy aging, most types of brain neurons are not lost in large numbers. In AD, however, where damage is widespread, many neurons stop functioning, lose connections with other neurons, and die because communication, metabolism, and repair are disrupted.

At first, AD typically destroys neurons and their connections in parts of the brain that control memory, including the entorhinal cortex and the hippocampus. It later attacks areas in the cerebral cortex responsible for language and reasoning. Eventually, many other areas of the brain are damaged, and a person with AD becomes helpless and unresponsive to the outside world.

#### **Characteristics of the AD Brain**

Many changes take place in the brain of a person with AD. Some of these changes can be observed under the microscope after death. The three abnormalities most evident in the brains of people who have died with AD are:

#### **Amyloid Plaques**

Found in the spaces between neurons, plaques consist of largely insoluble deposits of a protein fragment called beta-amyloid, which is generated from a protein called amyloid precursor protein (APP). Plaques also contain other proteins, remnants of degenerating neurons and glia, and other cellular material. Scientists used to think that amyloid plaques were the primary cause of the damage to neurons seen in AD. Now, however, many think that more soluble forms of beta-amyloid, seen earlier in the plaque formation process, may be the major culprits.

# **Neurofibrillary Tangles**

Found inside neurons, neurofibrillary tangles are abnormal aggregates of a protein called tau. Healthy neurons are internally supported in part by structures called microtubules, which help guide nutrients and molecules from the cell body to the end of the axon. Normally, tau binds to microtubules and helps stabilize them. In AD, tau undergoes abnormal chemical changes that cause it to disengage from microtubules and come together with other threads of tau, eventually forming neurofibrillary tangles. The microtubules disintegrate, and the neuron's transport system collapses. As with beta-amyloid, some scientists think that early soluble forms of abnormal tau may cause the most damage to neurons.

#### **Neuronal Repair and Cell Death**

Unlike most cells, which have a fairly short lifespan, nerve cells, which are generated in the fetus or a short time after birth, live a long time. Brain neurons can live for up to 100 years or longer. In an adult, when neurons die because of disease or injury, they are not usually replaced. Recent research, however, shows that in a few brain regions, new neurons can be born, even in the old brain.

To prevent their own death, living neurons must constantly maintain and remodel themselves. If cell cleanup and repair slows down or stops for any reason, the nerve cell cannot function well, and eventually it dies.

Alzheimer's disease disrupts each of the three processes that keep neurons healthy: communication, metabolism, and repair. This disruption causes certain nerve cells in the brain to stop working, lose connections with other nerve cells, and finally, die. The destruction and death of nerve cells causes the memory failure, personality changes, problems in carrying out daily activities, and other features of the disease.

### **Etiology**

More than 90 percent of AD cases occur in people older than 60. The development and pathology of this "late-onset" form of the disease are very similar to those of early-onset AD. The causes of late-onset AD are not yet completely understood, but they probably include a combination of genetic, environmental, and lifestyle factors. The importance of any one of these factors in increasing or decreasing the risk of developing AD may differ from person to person.

In some rare cases, people develop AD in their 40s or 50s. This form of the disease, called "early-onset" AD, often runs in families and is caused by a mutation in one of three genes that a person has inherited from a parent. The causes of other early-onset cases are not yet understood.

Much basic research in AD has focused on the genes that cause early-onset AD, and how mutations in these genes disrupt cellular function and lead to disease. Scientists hope that what they learn about early-onset AD can be applied to the late-onset form of the disease.

#### **Genetics**

## **Early-Onset Alzheimer's Disease**

Early-onset Alzheimer's disease occurs in people age 30 to 60. It is rare, representing less than 5 percent of all people who have Alzheimer's. Some cases of early-onset Alzheimer's have no known cause, but most cases are inherited, a type known as familial Alzheimer's disease (FAD).

Familial Alzheimer's disease is caused by any one of a number of different single-gene mutations on chromosomes 21, 14, and 1. Each of these mutations causes abnormal proteins to be formed. Mutations on chromosome 21 cause the formation of abnormal amyloid precursor protein (APP). A mutation on chromosome 14 causes abnormal presenilin 1 to be made, and a mutation on chromosome 1 leads to abnormal presenilin 2.

Scientists know that each of these mutations plays a role in the breakdown of APP, a protein whose precise function is not yet known. This breakdown is part of a process that generates harmful forms of amyloid plaques, a hallmark of the disease. A child whose mother or father carries a genetic mutation for FAD has a 50/50 chance of inheriting that mutation. If the mutation is in fact inherited, the child almost surely will develop FAD.

Critical research findings about early-onset Alzheimer's have helped identify key steps in the formation of brain abnormalities typical of Alzheimer's disease. They have also led to the development of imaging tests that show the accumulation of amyloid in the living brain. In addition, the study of Alzheimer's genetics has helped explain some of the variation in the age at which the disease develops.

Scientists are continuing this research through the Dominantly Inherited Alzheimer Network (DIAN), an international partnership to study families with a genetic mutation that causes early-onset Alzheimer's disease. By observing the biological changes that occur in these families long before symptoms appear, scientists hope to gain insight into how and why the disease develops in both its early- and late-onset forms. In addition, scientists are attempting to develop tests that will enable diagnosis of Alzheimer's before clinical signs and symptoms appear, as it is likely that early treatment will be critical as therapies become available.

### **Late-Onset Alzheimer's Disease**

Most cases of Alzheimer's are the late-onset form, which develops after age 60. The causes of late-onset Alzheimer's are not yet completely understood, but they likely include a combination of genetic, environmental, and lifestyle factors that influence a person's risk for developing the disease.

The single-gene mutations directly responsible for early-onset Alzheimer's disease do not seem to be involved in late-onset Alzheimer's. Researchers have not found a specific gene that causes the late-onset form of the disease. However, one genetic risk factor does appear to increase a person's risk of developing the disease. This increased risk is related to the apolipoprotein E (APOE) gene found on chromosome 19. APOE contains the instructions for making a protein that helps carry cholesterol and other types of fat in the bloodstream. APOE comes in several different forms, or alleles. Three forms— APOE  $\epsilon$ 2, APOE  $\epsilon$ 3, and APOE  $\epsilon$ 4—occur most frequently.

- APOE ε2 is relatively rare and may provide some protection against the disease. If Alzheimer's disease occurs in a person with this allele, it develops later in life than it would in someone with the APOE ε4 gene.
- APOE ε3, the most common allele, is believed to play a neutral role in the disease—neither decreasing nor increasing risk.
- APOE ε4 is present in about 25 to 30 percent of the population and in about 40 percent of all people with late-onset Alzheimer's. People who develop Alzheimer's are more likely to have an APOE ε4 allele than people who do not develop the disease.

Dozens of studies have confirmed that the APOE  $\epsilon 4$  allele increases the risk of developing Alzheimer's, but how that happens is not yet understood. These studies also help explain some of the variation in the age at which Alzheimer's disease develops, as people who inherit one or two APOE  $\epsilon 4$  alleles tend to develop the disease at an earlier age than those who do not have any APOE  $\epsilon 4$  alleles.

APOE ε4 is called a risk-factor gene because it increases a person's risk of developing the disease. However, inheriting an APOE ε4 allele does not mean

that a person will definitely develop Alzheimer's. Some people with one or two APOE  $\epsilon$ 4 alleles never get the disease, and others who develop Alzheimer's do not have any APOE  $\epsilon$ 4 alleles.

Using a relatively new approach called genome-wide association study (GWAS), researchers have identified a number of genes in addition to APOE  $\epsilon$ 4 that may increase a person's risk for late-onset Alzheimer's, including BIN1, CLU, PICALM, and CR1. Finding genetic risk factors like these helps scientists better understand how Alzheimer's disease develops and identify possible treatments to study.

# Lifestyle

Another area that is capturing an increasing amount of attention and interest is the possible influence of education, leisure, physical, and intellectually stimulating activities on the risk of developing AD. The interaction of genetic and lifestyle factors is also of interest. A number of studies over the past few years have provided intriguing hints that these activities may be linked to a reduced risk of AD, and they are consistent with what we know about other health benefits of lifelong physical and intellectual activity.

# **Cholesterol and Homocysteine**

A third exciting area of research is providing data about factors that may protect against or increase the risk of AD. In recent years, a number of studies have suggested a connection between AD and cholesterol in the blood. For example, the APOE-e4 allele is a variant of the APOE gene, which codes apolipoprotein E, a protein that helps to carry cholesterol in the blood. Test tube studies also have shown that blood cholesterol increases production of beta-amyloid from its APP precursor, and animal studies show a relationship between blood cholesterol and brain plaque levels in transgenic mice. Epidemiologic studies linking vascular risk factors to dementia have lent further support to this relationship. Many questions remain about the relationship between blood cholesterol and AD, but these intriguing findings have spurred new research and led scientists to hypothesize that drugs that lower blood cholesterol might also lower risk of developing dementia and AD.

#### Tau

In studying tau and what can go wrong, investigators have found that tau abnormalities are also central to other rare neurodegenerative diseases. These diseases, called tauopathies, include frontotemporal dementia, Pick's disease, supranuclear palsy, and corticobasal degeneration. They share a number of characteristics, but also each has distinct features that set them apart from each other and from AD. Characteristic signs and symptoms include changes in personality, social behavior, and language ability; difficulties in thinking and

making decisions; poor coordination and balance; psychiatric symptoms; and dementia. Recent advances include the discovery of mutations in the tau gene that cause one tauopathy called frontotemporal dementia with parkinsonism linked to chromosome 17 (FTDP-17). The development of several mouse models that produce tau tangles, will allow researchers to address the many questions that remain about these diseases.

# **Oxidative Damage from Free Radicals**

Another promising area of investigation relates to a longstanding theory of aging. This theory suggests that over time, damage from a kind of molecule called a free radical can build up in neurons, causing a loss in function. Free radicals can help cells in certain ways, such as fighting infection. However, too many can injure cells because they are very active and can readily change other nearby molecules, such as those in the neuron's cell membrane or in DNA. The resulting molecules can set off a chain reaction, releasing even more free radicals that can further damage neurons. This kind of damage is called oxidative damage. It may contribute to AD by upsetting the delicate machinery that controls the flow of substances in and out of the cell.

The brain's unique characteristics, including its high rate of metabolism and its long-lived cells, may make it especially vulnerable to oxidative damage over the lifespan. Some epidemiological and laboratory studies suggest that anti-oxidants from dietary supplements or food may provide some protection against developing AD. Other studies suggest that low calorie diets may protect against the development of AD by slowing down metabolic rates.

#### Inflammation

Another set of hints about the causes of AD points to inflammation in the brain. Because cells and compounds that are known to be involved in inflammation are found in AD plaques, some researchers think it may play a role in AD.

They disagree, though, on whether inflammation is a good or a bad thing. Some think it is harmful - that it sets off a vicious cycle of events that ultimately causes neurons to die. Evidence from many studies supports this idea.

Other scientists believe that some aspects of the inflammatory process may be helpful - that they are part of a healing process in the brain. For example, certain inflammatory processes may play a role in combating the accumulation of plaques. Many studies are now underway to examine the different parts of the inflammatory process more fully and their effects on AD.

#### **Aluminum**

Certain aluminum compounds have been found to be an important component of the neurological damage characteristics of Alzheimer's Disease. Much research over the last decade has focused on the role of aluminum in the development of this disease. At this point, its role is still not clearly defined. Since AD is a chronic disease which may take a long time to develop, long-term exposure is the most important measure of intake. Long-term exposure is easiest to estimate for drinking water exposures. Epidemiological studies attempting to link AD with exposures in drinking water have been inconclusive and contradictory. Thus, the significance of increased aluminum intake with regard to onset of AD has not been determined.

Aluminum is one of the most abundant elements found in the environment. Therefore, human exposure to this metal is common and unavoidable. However, intake is relatively low because this element is highly insoluble in many of its naturally occurring forms. The significance of environmental contact with aluminum is further diminished by the fact that less than 1% of that taken into the body orally is absorbed from the gastrointestinal tract.

Over the last few years, there has been concern about the exposures resulting from leaching of aluminum from cookware and beverage cans. However, as a general rule, this contributes a relatively small amount to the total daily intake. Aluminum beverage cans are usually coated with a polymer to minimize such leaching. Leaching from aluminum cookware becomes potentially significant only when cooking highly basic or acidic foods.

#### Copper and Zinc

Additional work in beta-amyloid has built on earlier studies indicating that the amount of copper and zinc is increased in the cortex of brains from individuals who have died of AD. These metals are concentrated in beta-amyloid plaques. Although controversial, some scientists believe that beta-amyloid possesses binding sites for copper and zinc that enhance the resistance of beta-amyloid to breakdown by enzymes and encourage its tendency to clump together to form plaques.

#### **Symptomology**

As Alzheimer's disease makes inroads into memory and mental abilities, it also begins to change a person's emotions and behaviors. Between 70 to 90 percent of people with Alzheimer's disease eventually develop one or more behavioral symptoms. These include sleeplessness, wandering and pacing, aggression, agitation, anger, depression, and hallucinations and delusions. Some of these

symptoms often become worse in the evening, a phenomenon called "sundowning," or during daily routines, especially bathing.

Unlike a stroke, in which damage to part of the brain occurs all at once, the damage of Alzheimer's disease spreads slowly over time and affects many different parts of the brain. Even small tasks require the brain to engage in a complex process that can involve more than one region of the brain. If this process is disrupted, the person may not be able to do the task or may act in a strange or inappropriate way.

Behavioral symptoms are one of the hardest aspects of the disease for families and other caregivers to deal with. They are emotional and upsetting. They are also a visible sign of the terrible change that has taken place in the person with AD.

# 10 Warning Signs of Alzheimer's:

#### **Memory loss**

Forgetting recently learned information is one of the most common early signs of dementia. A person begins to forget more often and is unable to recall the information later. What's normal? Occasionally forgetting names or appointments.

# Difficulty performing familiar tasks

People with dementia often find it hard to plan or complete everyday tasks. Individuals may lose track of the steps involved in preparing a meal, placing a telephone call or playing a game. What's normal? Occasionally forgetting why you came into a room or what you planned to say.

#### **Problems with language**

People with Alzheimer's disease often forget simple words or substitute unusual words, making their speech or writing hard to understand. They may be unable to find the toothbrush, for example, and instead ask for "that thing for my mouth." What's normal? Sometimes having trouble finding the right word.

#### Disorientation to time and place

People with Alzheimer's disease can become lost in their own neighborhood, forget where they are and how they got there, and not know how to get back home. What's normal? Forgetting the day of the week or where you were going.

### Poor or decreased judgment

Those with Alzheimer's may dress inappropriately, wearing several layers on a warm day or little clothing in the cold. They may show poor judgment, like giving away large sums of money to telemarketers. What's normal? Making a questionable or debatable decision from time to time.

# Problems with abstract thinking

Someone with Alzheimer's disease may have unusual difficulty performing complex mental tasks, like forgetting what numbers are for and how they should be used. What's normal? Finding it challenging to balance a checkbook.

# Misplacing things

A person with Alzheimer's disease may put things in unusual places: an iron in the freezer or a wristwatch in the sugar bowl. What's normal? Misplacing keys or a wallet temporarily.

# Changes in mood or behavior

Someone with Alzheimer's disease may show rapid mood swings – from calm to tears to anger – for no apparent reason. What's normal? Occasionally feeling sad or moody.

# **Changes in personality**

The personalities of people with dementia can change dramatically. They may become extremely confused, suspicious, fearful or dependent on a family member. What's normal? People's personalities do change somewhat with age.

#### Loss of initiative

A person with Alzheimer's disease may become very passive, sitting in front of the TV for hours, sleeping more than usual or not wanting to do usual activities. What's normal? Sometimes feeling weary of work or social obligations.

Alzheimer's disease develops slowly and causes changes in the brain long before there are obvious changes in a person's memory, thinking, use of words or behavior. Stages and changes the person will go through are outlined below.

# **Stages of Alzheimer's Disease**

Although the course of Alzheimer's Disease is not the same in every patient, symptoms seem to develop over the same general stages.

Alzheimer's disease typically develops slowly and causes a gradual decline in cognitive abilities, usually over a span of seven to 10 years. It eventually affects all brain functions, including memory, movement, language, behavior, judgment and abstract reasoning. Not everyone will experience the same symptoms or progress at the same rate. The rate of progression varies widely among individuals. For some, severe dementia occurs within five years of diagnosis. For others, it can take more than a decade. On average, people with Alzheimer's live for eight to 10 years after diagnosis. Some live as long as 20 years.

While each individual is different, the progression of his or her disease can be roughly divided into mild, moderate, and severe stages. In order to identify

symptoms and to assist in identifying what services individuals and caregivers may need, the Alzheimer's Association provides the following stage categories:

# Stage 1: No impairment (normal function)

Unimpaired individuals experience no memory problems and none are evident to a health care professional during a medical interview.

# Stage 2: Very mild cognitive decline (may be normal age-related changes or earliest signs of Alzheimer's disease)

Individuals may feel as if they have memory lapses, especially in forgetting familiar words or names or the location of keys, eyeglasses or other everyday objects. But these problems are not evident during a medical examination or apparent to friends, family or co-workers.

# Stage 3: Mild cognitive decline (Early-stage Alzheimer's can be diagnosed in some, but not all, individuals with these symptoms)

Friends, family or co-workers begin to notice deficiencies. Problems with memory or concentration may be measurable in clinical testing or discernible during a detailed medical interview.

#### Common difficulties include:

- Word- or name-finding problems noticeable to family or close associates.
- Decreased ability to remember names when introduced to new people.
- Performance issues in social or work settings noticeable to family, friends or co-workers.
- Reading a passage and retaining little material.
- Losing or misplacing a valuable object.
- Decline in ability to plan or organize.

# Stage 4: Moderate cognitive decline (Mild or early-stage Alzheimer's disease)

At this stage, a careful medical interview detects clear-cut deficiencies in the following areas:

- Decreased knowledge of recent occasions or current events.
- Impaired ability to perform challenging mental arithmetic-for example, to count backward from 75 by 7s.
- Decreased capacity to perform complex tasks, such as planning dinner for guests, paying bills and managing finances.
- Reduced memory of personal history.
- The affected individual may seem subdued and withdrawn, especially in socially or mentally challenging situations.

# **Stage 5: Moderately severe cognitive decline (Moderate or mid-stage AD)** Major gaps in memory and deficits in cognitive function emerge. Some assistance with day-to –day activities becomes essential. At this stage, individuals may:

- Be unable during a medical interview to recall such important details as their current address, their telephone number or the name of the college or high school from which they graduated.
- Become confused about where they are or about the date, day of the week or season.
- Have trouble with less challenging mental arithmetic; for example, counting backward from 40 by 4s or from 20 by 2s.
- Need help choosing proper clothing for the season or the occasion.
- Usually retain substantial knowledge about themselves and know their own name and the names of their spouse or children.
- Usually require no assistance with eating or using the toilet.

# Stage 6: Severe cognitive decline (Moderately, severe, or mid-stage AD).

Memory difficulties continue to worsen, significant personality changes may emerge and affected individuals need extensive help with customary daily activities.

At this stage, individuals may:

- Lose most awareness of recent experiences and events as well as of their surroundings.
- Recollect their personal history imperfectly, although they generally recall their own name.
- Occasionally forget the name of their spouse or primary caregiver but generally can distinguish familiar from unfamiliar faces.
- Need help getting dressed properly; without supervision, may make such errors as putting pajamas over daytime clothes or shoes on wrong feet.
- Experience disruptions of their normal sleep/waking cycle.
- Need help with handling details of toileting (flushing toilet, wiping and disposing of tissue properly).
- Have increasing episodes of urinary or fecal incontinence.
- Experience significant personality changes and behavioral symptoms, including suspiciousness and delusions (for example, believing that their caregiver is an impostor); hallucinations (seeing or hearing things that are not really there); or compulsive, repetitive behaviors such as handwringing or tissue shredding.
- Tend to wander and become lost.

### Stage 7: Very severe cognitive decline (Severe or late-stage AD)

This is the final stage of the disease when individuals lose the ability to respond to their environment, the ability to speak and, ultimately, the ability to control movement.

#### Alzheimer's Disease

- Frequently individuals lose their capacity for recognizable speech, although words or phrases may occasionally be uttered.
- Individuals need help with eating and toileting and there is general incontinence of urine.
- Individuals lose the ability to walk without assistance, then the ability to sit without support, the ability to smile, and the ability to hold their head up.
- Reflexes become abnormal and muscles grow rigid.
- Swallowing is impaired.

# **Diagnosis**

Currently, experienced clinicians can diagnose AD with up to 90 percent accuracy. However, we are still some distance from the ultimate goal - a reliable, valid, inexpensive, and early diagnostic marker.

Early diagnosis has several advantages. For example, many conditions cause symptoms that mimic those of Alzheimer's disease. Finding out early that the problem isn't AD but is something else, can spur people into getting treatment for the real condition. For the small percentage of dementias that are treatable or even reversible, early diagnosis increases the chances of successful treatment.

Even when the cause of the dementia turns out to be Alzheimer's disease, it's good to find out sooner rather than later. One benefit is medical. The drugs now available to treat AD can help some people maintain their mental abilities for months to years, though they do not change the underlying course of the disease.

Other benefits are practical. The sooner the person with AD and family know, the more time they have to make future living arrangements, handle financial matters, establish a durable power of attorney, deal with other legal issues, create a support network, or even make plans to join a research study. Being able to participate for as long as possible in making decisions about the present and future, is important to many people with AD.

# **New Diagnostic Guidelines**

In 2011 three expert international workgroups convened by the Alzheimer's Association and the National Institute on Aging (NIA) issued the first new criteria and guidelines to diagnose Alzheimer's disease in nearly three decades. The new guidelines update, refine and broaden widely used guidelines published in 1984 by the Alzheimer's Association (then known as the Alzheimer's Disease and Related Disorders Association) and the National Institute of Neurological and Communicative Disorders and Stroke (now known as the National Institute of Neurological Disorders and Stroke).

#### Alzheimer's Disease

According to the Alzheimer's Association the new diagnostic criteria and guidelines include the following key elements:

- Updates to widely used existing guidelines for Alzheimer's disease originally established in 1984 by the National Institute of Neurological Disorders and Stroke (NINDS) and the Alzheimer's Association.
- Refinements to existing guidelines for diagnosing mild cognitive impairment (MCI). People with MCI experience a decline in memory, reasoning or visual perception that's measurable and noticeable to themselves or to others, but not severe enough to be diagnosed as Alzheimer's or another dementia. The new guidelines formalize an emerging consensus that everyone who eventually develops Alzheimer's experiences this stage of minimal but detectable impairment, even though it's not currently diagnosed in most people. However, not everyone with MCI eventually develops Alzheimer's, because MCI may also occur for other reasons. The guidelines designate the condition of minimal impairment preceding Alzheimer's as "MCI due to Alzheimer's disease," and define four levels of certainty for arriving at this diagnosis.
- Expansion of the conceptual framework for thinking about Alzheimer's
  disease to include a "preclinical" stage characterized by signature
  biological changes (biomarkers) that occur years before any disruptions in
  memory, thinking or behavior can be detected. The new guidelines do not
  yet specify which biomarkers should be considered signatures of
  preclinical Alzheimer's. Instead, they propose a research agenda that
  builds on promising preliminary data emerging from recent studies,
  including the federally funded Alzheimer's Disease Neuroimaging Initiative
  (ADNI). Promising investigational biomarkers include brain imaging
  strategies and certain proteins in spinal fluid.
- Establishment of a framework for eventually adding biomarker benchmarks to the diagnosis of Alzheimer's disease in all of its stages. The guidelines for MCI due to Alzheimer's disease include specific biomarkers for use in research settings, with the expectation that these recommendations are a work in progress that will evolve as knowledge advances. The guidelines for dementia due to Alzheimer's disease also propose a research agenda to increase diagnostic certainty by incorporating biomarkers into the diagnosis. Understanding signature biomarkers may reveal how these benchmarks change over time, enabling more precise and clinically useful characterization of the full spectrum of Alzheimer's. These insights may pave the way for more effective treatments targeted at signature pathological processes at work in specific stages.

Clifford R. Jack Jr., et al. "Introduction to the Recommendations from the National Institute on Aging and the Alzheimer's Association Workgroups on Diagnostic Guidelines for Alzheimer's Disease."

Guy M. McKhann and David S. Knopman, et al. "The Diagnosis of Dementia Due to Alzheimer's Disease: Recommendations from the National Institute on Aging and the Alzheimer's Association Workgroup."

Marilyn S. Albert, et al. "The Diagnosis of Mild Cognitive Impairment Due to Alzheimer's Disease: Recommendations from the National Institute on Aging and Alzheimer's Association Workgroup."

Reisa A. Sperling, et al. "Toward Defining the Preclinical Stages of Alzheimer's Disease: Recommendations from the National Institute on Aging and the Alzheimer's Association Workgroup."

# **Diagnostic Tools**

A definitive diagnosis of Alzheimer's disease is still only possible after death, during an autopsy, when the plaques and tangles can actually be seen. But with the tools now available, experienced clinicians can be pretty confident about making an accurate diagnosis in a living person. A comprehensive assessment for Alzheimer's disease usually includes all of the following.

- **Detailed Patient History** How and when symptoms developed. The patient's and his or her family's overall medical condition and history. An assessment of the patient's emotional state and living environment.
- Interview of family members or close friends People close to the patient can provide valuable insights into how behavior and personality have changed; many times, family and friends know something is wrong even before changes are evident on tests.
- Physical and neurological examinations and laboratory tests Blood and other medical tests help determine neurological functioning and identify possible non-AD causes of dementia.

# **Biological Markers and Oxidative Stress**

Scientists are also trying to discover whether biological markers exist that could indicate early changes in the brain associated with AD. Understanding more about these markers - what they are, how they function, and how and when their levels change - will help investigators answer questions about the cause and development of AD and may lead one day to treatments to delay or prevent the onset of the disease.

# **Neuroimaging**

Investigators are continuing to use neuroimaging techniques, such as magnetic resonance imaging (MRI) and positron emission tomography (PET), to assess whether it is possible to measure aspects of brain structure or function that will identify those people who are at risk of AD before they develop the symptoms of the disease. Over the past few years, results from a number of promising longitudinal neuroimaging studies have been published. These studies have expanded our understanding of the potential usefulness of imaging techniques for research and diagnostic purposes as well as increased our knowledge about early AD changes in the brain.

# Neuropsychology

Several research teams have hypothesized that performance on specific cognitive tests might predict whether an individual will develop AD.

Guidelines recommend that clinicians use general cognitive screening instruments, such as the Mini-Mental State Examination, and other neuropsychological tests to evaluate and monitor patients with mild cognitive impairment. These tests may help clinicians assess the degree of cognitive impairment and help them detect signs that might indicate the development of dementia.

The Mini Mental State Examination (MMSE) - is the most commonly used test for complaints of memory problems or when a diagnosis of dementia is being considered.

The MMSE is a series of questions and tests, each of which scores points if answered correctly. If every answer is correct, a maximum score of 30 points is possible. People with Alzheimer's disease generally score 26 points or less.

#### **Section 1: Orientation**

The first 10 points are gained for giving the correct date and location. For example:

- What is the day of the week?
- What year was last year?
- What is the street name?
- What building are we in?

# Section 2: Memory (part 1)

The first part of the memory test evaluates short-term memory. The patient is given the names of three objects to remember - table, ball and pen, for example. They are then be asked to repeat the three names, scoring 1 point for each object correctly recalled (3 points maximum).

### Section 3: Attention and calculation

The next part of the MMSE tests the ability to concentrate on a tricky task.

For example, the patient may be asked to count backwards by 5 starting at 50. One point is given for each correct subtraction, with a maximum of 5 points.

# Section 4: Memory (part 2)

The patient is asked to recall the three items from Section 2. The attention and calculation section may have been quite a stressful experience, so this can be tricky. One point is given for each correctly recalled object.

# Section 5: Language, writing and drawing

The final part of the test makes an assessment of spoken and written language, and the ability to write and copy.

The person being tested is shown two everyday items - a hammer and a crayon, for example - and asked to name them. One point is scored for each correct answer.

The patient is then asked to say aloud a tongue-twister sentence such as 'Pass the peas please'. One point is scored for correctly repeating the sentence.

The patient is then given a piece of paper, and asked to carry out a threestep process:

For example:

- 'Take this paper in your hand' (1 point);
- 'Fold it in half' (1 point);
- 'Place it on this chair' (1 point).

One point is gained for each correctly completed step.

A card is then shown with an instruction for a simple task - 'Clap your hands'. One point is scored for a correct response.

The next stage of the test is to write a sentence on a piece of paper. The sentence needs to make sense. One point is scored for an acceptable sentence.

Finally, the ability to copy a design of two intersecting shapes is assessed. One point is awarded for correctly copying it. All angles on both figures must be present, and the figures must have one overlapping angle.

#### **APOE Testing**

A blood test is available that can identify which apoE alleles a person has. However, because the apoE4 gene is only a risk factor for AD, this blood test cannot tell whether a person will develop AD or not. In diagnosing AD, apoE testing is not a common practice. Instead of a yes or no answer, the best

information a person can get from this genetic test for apoE is maybe or maybe not. Although some people want to know whether they will get AD later in life, this type of prediction is not yet possible. In fact, some researchers believe that screening measures may never be able to predict AD with 100 percent accuracy. In a research setting, apoE testing may be used to identify study volunteers who may be at a higher risk of getting AD. In this way, researchers can look for early brain changes in some patients. This test also helps researchers compare the effectiveness of treatments for patients with different apoE profiles. Most researchers believe that the apoE test is useful for studying AD risk in large groups of people but not for determining one person's individual risk. Predictive screening in otherwise healthy people will be useful if an accurate/reliable test is developed and effective ways to treat or prevent AD are available.

#### **Treatment**

#### **Medications**

Only a few medications have been approved by the U.S. Food and Drug Administration (FDA) to help control the cognitive loss that characterizes AD.

#### **Cholinesterase Inhibitors**

The first FDA-approved Alzheimer medications were cholinesterase inhibitors. Three of these medications are commonly prescribed— donepezil (Aricept®), rivastigmine (Exelon®), and galantamine (Reminyl®).

These drugs are prescribed for the treatment of mild to moderate AD.

Cholinesterase inhibitors are designed to enhance memory and other cognitive functions by influencing certain chemical activities in the brain. Acetylcholine is a chemical messenger in the brain that scientists believe is important for the function of brain cells involved in memory, thought, and judgment. Acetylcholine is released by one brain cell to transmit a message to another. Once a message is received, various enzymes, including one called acetylcholinesterase, break down the chemical messenger for reuse.

In the Alzheimer-afflicted brain, the cells that use acetylcholine are damaged or destroyed, resulting in lower levels of the chemical messenger. A cholinesterase inhibitor is designed to stop the activity of acetylcholinesterase, thereby slowing the breakdown of acetylcholine. By maintaining levels of acetylcholine, the drug may help compensate for the loss of functioning brain cells. Galantamine also appears to stimulate the release of acetylcholine and to strengthen the way that certain receptors on message-receiving nerve cells respond to it.

The drugs maintain some patients' abilities to carry out everyday activities and may slow down symptoms related to thinking, memory, or speaking skills. They

#### Alzheimer's Disease

also may help with certain behavioral symptoms. However, they do not stop or reverse AD and appear to help patients only for months to a few years.

#### Memantine

Another type of medication, memantine (Namenda<sup>®</sup>), is prescribed to treat moderate to severe AD symptoms. This drug appears to work by blocking receptors for glutamate, another neurotransmitter involved in memory function. Studies in animals suggest that memantine may have disease-modifying effects, although this has not yet been demonstrated in humans.

Memantine is classified as an uncompetitive low-to- moderate affinity N-methyl-D-aspartate (NMDA) receptor antagonist. It appears to work by regulating the activity of glutamate, one of the brain's specialized messenger chemicals involved in information processing, storage, and retrieval. Glutamate plays an essential role in learning and memory by triggering NMDA receptors to allow a controlled amount of calcium to flow into a nerve cell, creating the chemical environment required for information storage.

Excess glutamate, overstimulates NMDA receptors to allow too much calcium into nerve cells, leading to disruption and death of cells. Memantine may protect cells against excess glutamate by partially blocking NMDA receptors. Memantine's action differs from the mechanism of the cholinesterase inhibitors that were previously approved in the United States for treatment of Alzheimer symptoms. It is hoped that this drug will help maintain personal care functions and assist in behavior management longer.

#### **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. Some additional differences among these medications are summarized in the table located on following page.

# Alzheimer's Disease

DRUG NAME	DRUG TYPE AND TREATMENT	MANUFACTURER'S RECOMMENDED DOSAGE	COMMON SIDE EFFECTS	POSSIBLE DRUG INTERACTIONS
Namenda® (memantine)  Blocks the toxic effects associated with excess glutamate and regulates glutamate activation.	N-methyl D-aspartate (NMDA) antagonist prescribed to treat symptoms of moderate to severe AD	Increase to 10 mg/day (5 mg twice a day), 15 mg/day (5 mg and 10 mg as separate doses), and 20 mg/day (10 mg twice a day) at minimum of one week intervals if well tolerated.	Dizziness, headache, constipation, confusion	Other NMDA antagonist medications, including amantadine, an antiviral used to treat the flu, dextromethorphan, prescribed to relieve coughs due to colds or flu, and ketamine, sometimes used as an anesthetic, have not been systematically evaluated and should be used with caution in combination with this medications.
Reminyl® (galantamine)  Prevents the breakdown of acetylcholine and stimulates nicotinic receptors to release more acetylcholine in the brain.	Cholinesterase inhibitor prescribed to treat symptoms of mild to moderate AD	<ul> <li>4mg, twice a day (8mg/day)</li> <li>Increase by 8mg/day after 4 weeks to 8mg, twice a day (16mg/day)if well tolerated.</li> <li>After another 4 weeks, increase to 12mg, twice a day (24mg/day) if well tolerated.</li> </ul>	Nausea, vomiting, diarrhea, weight loss	Some antidepressants such as paroxetine, amitriptyline, fluoxetine, fluoxetine, and other drugs with anticholinergic action may cause retention of excess Reminyl in the body, leading to complications; NSAIDs should be used with caution in combination with this medication.*
Exelon® (rivastigmine)  Prevents the breakdown of acetylcholine and butyrylcholine (a brain chemical similar to acetylcholine) in the brain.	Cholinesterase inhibitor prescribed to treat symptoms of mild to moderate AD	<ul> <li>1.5mg, twice a day (3mg/day)</li> <li>Increase by 3mg/day every 2 weeks to 6mg, twice a day (12mg/day) if well tolerated.</li> </ul>	Nausea, vomiting, weight loss, upset stomach, muscle weakness	None observed in laboratory studies; NSAIDs should be used with caution in combination with this medication.*
Aricept® (donepezil)  Prevents the breakdown of acetylcholine in the brain.	Cholinesterase inhibitor prescribed to treat symptoms of mild to moderate AD	<ul> <li>5mg, once a day</li> <li>Increase after 4-6         weeks to 10mg,         once a day if well         tolerated.</li> </ul>	Nausea, diarrhea, vomiting	None observed in laboratory studies; NSAIDs should be used with caution in combination with this medication.*

It has become clear that there probably isn't a "magic bullet" that will, by itself, prevent or cure AD. However, scientists are identifying a number of interventions that can be used to reduce risk and treat the disease. Today, it is estimated that the National Institute on Aging, other NIH Institutes, and private industry are conducting clinical trials on around 30 compounds that may be active against AD.

#### **Other Treatments**

In addition to these medications, physicians may use other drugs and non-drug approaches to treat behavioral and psychiatric problems associated with AD. These problems include agitation, verbal and physical aggression, wandering, depression, sleep disturbances, and delusions. (It is important to note, however, that since no drugs are specifically approved by the FDA to treat behavioral or psychiatric symptoms in dementia, this practice constitutes "off-label" usage.)

#### **Nerve Growth Factor**

Nerve growth factor (NGF) is one of several growth factors in the body that maintain the health of neurons. NGF also promotes the growth of axons and dendrites, the neuron branches that connect with other neurons and that are essential in nerve cells' ability to communicate. Studies have turned up a number of clues that link NGF to the neurons that use acetylcholine as a neurotransmitter, so researchers have been eager to see what happens when NGF is added to aging brain tissue.

# **Nutritional Supplements**

Research has shown that vitamin E slows the progress of some consequences of AD by about 7 months. Scientists now are studying vitamin E to learn whether it can prevent or delay AD in patients with mild cognitive impairment (MCI).

The dietary supplement *Ginkgo biloba*, enthusiastically promoted by many to improve cognitive abilities, has been found to be ineffective in reducing the development of dementia and Alzheimer's disease in older people, according to a study published in the *Journal of the American Medical Association* 

Fish oil, when combined with epigallocatechin-3-gallate (EGCG—a polyphenol and antioxidant found in green tea), may affect chemical processes in the brain associated with Alzheimer's disease, according to a study published in *Neuroscience Letters*.

Omega-3 fatty acid docosahexaenoic acid (DHA); and curcumin, a component of turmeric, may affect Alzheimer's related chemical processes in the brain, according to a study reported in *The Journal of Neuroscience*. Researchers, studying mice, found that a high-fat diet increased Alzheimer's disease—related chemical processes in the brain, and that fish oil and curcumin counteracted this effect. DHA and curcumin also protected cognitive performance for mice on a high-fat diet.

# Other Approaches

Finally, a number of clinical trials are focusing on the earliest stages of the disease process. For example, scientists are developing drugs that prevent enzymes from clipping beta-amyloid out from APP. Others are working on ways to stop beta-amyloid from clumping together into plaques. Teams of investigators are also studying certain enzymes that seem to be able to break beta-amyloid into pieces after it is released from cells but before it has a chance to form into plaques. Still other scientists are exploring the role of neurotransmitter systems other than acetylcholine, such as glutamate. One especially active area of research involves the possibility that a vaccine might be able to stimulate the immune system into getting rid of plaques once they have formed, stopping beta-amyloid and plaque buildup, or even getting rid of plaques once they have formed.

# Caregiving

Currently, the vast majority of individuals living with Alzheimer's disease are cared for at home by family members. In fact, an estimated 25–29 percent of caregivers of persons age 50 or older (8.5–9.8 million caregivers) provide help to someone with a cognitive impairment, a memory problem, or a disorder such as Alzheimer's disease or another form of dementia.

Caring for a person with Alzheimer's disease at home is a difficult task and can become overwhelming at times. Each day brings new challenges as the caregiver copes with changing levels of ability and new patterns of behavior. As the disease gets worse, people living with Alzheimer's disease often need more and more care.

In addition to understanding the challenges of caring for someone living with Alzheimer's disease, it is also important to note that caregiving has positive aspects. It is usually undertaken willingly and may bring personal fulfillment to family caregivers, such as satisfaction from helping a family member or friend, development of new skills and improved family relationships. Caregiving is receiving increased attention as an important public health issue.

Perhaps one of the greatest costs of Alzheimer's disease is the physical and emotional toll on family, caregivers, and friends. The changes in a loved one's personality and mental abilities; the need to provide constant, loving attention for years on end; and the demands of bathing, dressing, and other caregiving duties can be hard to bear. Many caregivers must assume new and unfamiliar roles in the family and these changes can be both difficult and sad. Not surprisingly, caregivers of people with dementia spend significantly more time on caregiving tasks than do caregivers of people with other types of illnesses.

Although research on caregiver support is still in its early days, a lot is already known about the unique aspects of caregivers' personalities and situations.

Certain characteristics seem to make some caregivers more vulnerable to the physical and emotional stresses associated with dementia care. These characteristics include being a male spouse, having few breaks from caregiving responsibilities, and having preexisting illnesses.

Caregiver research is also beginning to tease out characteristics of support programs that might be most useful for particular groups of caregivers. For example, peer support programs that link caregivers with trained volunteers who also have been dementia caregivers appear to help. These programs are especially good for caregivers whose social support networks are weak or who are in very stressful situations. Other research has confirmed that the information and problem-solving needs of caregivers evolve over time as the person with AD changes. Support programs can respond by offering services and information geared to different stages of the disease.

One of the most difficult decisions that many families face is whether to place a loved one with Alzheimer's disease in a nursing home or other type of care facility. Once this decision is made, families must decide what type of care is best for the person and the family.

# Caring for a Person with AD

The following are some of the challenges faced by AD caregivers:

# Physical effort and time commitment

Help with bathing, eating, dressing, and other activities of daily living take a lot of time. As the disease progresses, the need for this kind of help increases. Behavior problems and safety concerns mean that the caregiver is always "on duty," even when not actively helping the person.

#### Financial costs

The costs of care vary, but can be high depending on whether the person is cared for at home or in a residential care setting and how much help the caregiver has. Many caregivers give up their jobs or cut back on their work hours and this also has financial implications.

### Psychological loss

Caregivers often experience a profound sense of loss as the disease slowly takes their husband, wife, parent, or friend. The relationship as it once was gradually ends and plans for the future must be radically changed. Caregivers must come to terms with "the long goodbye."

# Negative effects of being a caregiver

- Employment complications
- Emotional distress
- Fatigue and poor physical health
- Social isolation

- Family conflict
- Less time for leisure, self, and other family members

# Positive effects of being a caregiver

- A new sense of purpose or meaning in life
- Fulfillment of a lifelong commitment to a spouse
- An opportunity to give back to a parent some of what the parent has given to them
- Renewal of religious faith
- Closer ties with people through new relationships or stronger existing relationships

#### 9 R's in Alzheimer's Care

- 1. Routine will help someone with AD anticipate what to do next and what is expected of him or her. Routine sets boundaries that feel secure.
- 2. Rituals or life-long habits should be maintained. Do not try to break old habits. If the person has taken a walk after breakfast every day for thirty years, don't try to make them stop now.
- 3. Reassess physical health if there is a sudden behavior change. A person with AD will become more confused, agitated or hostile when a physical complaint develops.
- 4. Redirect or distract the person away from repetitive thought patterns. For example, if the person with AD is asking to go home, say, "In a little while, but first how about a cup of tea and a cookie?" This kind of suggestion can divert the person's thoughts, at least for a little while.
- 5. Real questions may be difficult to decipher if the person with AD has difficulty communicating. Increased agitation, for instance, could be his or her way of telling you a trip to the restroom is needed. Learn to read body language.
- 6. Reconsider the behavior. Is it really a problem, or is it just upsetting to you? Does it present a danger to the person with AD? If the person talks to his or her reflection in a mirror as if it was another person, is that a real problem? Does it please the person with AD or does it upset or frighten him or her?
- 7. Respect the person with AD. Laugh with, not at the person. Never talk about the person as if he or she is not there. And remember, the person with AD will know how you feel by your body language and tone of voice.
- 8. Reassure the person with AD often that you love and will care for him or her. A gentle pat, stroking the hair, or holding a hand all say, "I care for you."
- 9. Remember the person with AD is still a human being. Treat him or her the way you would want to be treated if you were suffering from dementia.

#### Communication

Trying to communicate with a person who has AD can be a challenge. Both understanding and being understood may be difficult.

- Choose simple words and short sentences and use a gentle, calm tone of voice.
- Avoid talking to the person with AD like a baby or talking about the person as if he or she weren't there.
- Minimize distractions and noise--such as the television or radio--to help the person focus on what you are saying.
- Call the person by name, making sure you have his or her attention before speaking.
- Allow enough time for a response. Be careful not to interrupt.
- If the person with AD is struggling to find a word or communicate a thought, gently try to provide the word he or she is looking for.
- Try to frame questions and instructions in a positive way.

# **Activities of Daily Living**

## Bathing

While some people with AD don't mind bathing, for others it is a frightening, confusing experience. Advance planning can help make bath time better for both of you.

- Plan the bath or shower for the time of day when the person is most calm and agreeable. Be consistent. Try to develop a routine.
- Respect the fact that bathing is scary and uncomfortable for some people with AD. Be gentle and respectful. Be patient and calm.
- Tell the person what you are going to do, step by step, and allow him or her to do as much as possible.
- Prepare in advance. Make sure you have everything you need ready and in the bathroom before beginning. Draw the bath ahead of time.
- Be sensitive to the temperature. Warm up the room beforehand if necessary and keep extra towels and a robe nearby. Test the water temperature before beginning the bath or shower.
- Minimize safety risks by using a handheld showerhead, shower bench, grab bars, and nonskid bath mats. Never leave the person alone in the bath or shower.
- Try a sponge bath. Bathing may not be necessary every day. A sponge bath can be effective between showers or baths.

# **Dressing**

Getting dressed presents a series of challenges for someone who has AD: choosing what to wear, getting some clothes off and other clothes on, and struggling with buttons and zippers. Minimizing the challenges may make a difference.

- Try to have the person get dressed at the same time each day so he or she will come to expect it as part of the daily routine.
- Encourage the person to dress himself or herself to whatever degree possible. Plan to allow extra time so there is no pressure or rush.
- Allow the person to choose from a limited selection of outfits. If he
  or she has a favorite outfit, consider buying several identical sets.
- Arrange the clothes in the order they are to be put on to help the person move through the process.
- Provide clear, step-by-step instructions if the person needs prompting.
- Choose clothing that is comfortable, easy to get on and off, and easy to care for. Elastic waists and Velcro enclosures minimize struggles with buttons and zippers.

# **Eating**

Eating can be a challenge. Some people with AD want to eat all the time, while others have to be encouraged to maintain a good diet.

- Ensure a quiet, calm atmosphere for eating. Limiting noise and other distractions may help the person focus on the meal.
- Provide a limited number of choices of food and serve small portions. You may want to offer several small meals throughout the day in place of three larger ones.
- Use straws or cups with lids to make drinking easier.
- Substitute finger foods if the person struggles with utensils. Using a bowl instead of a plate also may help.
- Have healthy snacks on hand. To encourage eating, keep the snacks where they can be seen.
- Visit the dentist regularly to keep mouth and teeth healthy.

#### Activities

Finding activities that the person with AD can do and is interested in can be a challenge. Building on current skills generally works better than trying to teach something new.

- Don't expect too much. Simple activities often are best, especially when they use current abilities.
- Help the person get started on an activity. Break the activity down into small steps and praise the person for each step he or she completes.
- Watch for signs of agitation or frustration with an activity. Gently help or distract the person to something else.
- Incorporate activities the person seems to enjoy into your daily routine and try to do them at a similar time each day.

#### Incontinence

As the disease progresses, many people with AD begin to experience incontinence. Incontinence can be upsetting to the person and difficult for the caregiver. Sometimes incontinence is due to physical illness, so be sure to discuss it with the person's doctor.

- Have a routine for taking the person to the bathroom and stick to it as closely as possible. For example, take the person to the bathroom every 3 hours or so during the day. Don't wait for the person to ask.
- Watch for signs that the person may have to go to the bathroom, such as restlessness or pulling at clothes. Respond quickly.
- Be understanding when accidents occur. Stay calm and reassure the person if he or she is upset. Try to keep track of when accidents happen to help plan ways to avoid them.
- To help prevent nighttime accidents, limit certain types of fluids-such as those with caffeine--in the evening.
- Always have an extra set of clothing available in case of an accident.

# **Sleep Problems**

For the exhausted caregiver, sleep can't come too soon. For many people with AD, however, nighttime may be a difficult time. Getting the person to go to bed and stay there may require some advance planning.

- Set a quiet, peaceful tone in the evening to encourage sleep. Keep the lights dim, eliminate loud noises, even play soothing music if the person seems to enjoy it.
- Try to keep bedtime at a similar time each evening. Developing a bedtime routine may help.
- Encourage exercise during the day and limit daytime napping.
- Restrict access to caffeine late in the day.

• Use night lights in the bedroom, hall, and bathroom if the darkness is frightening or disorienting.

#### **Exercise**

Incorporating exercise into the daily routine has benefits for both the person with AD and the caregiver. Not only can it improve health, but it also can provide a meaningful activity for both of them to share.

- Think about what kind of physical activities both enjoy, perhaps walking, swimming, tennis, dancing, or gardening. Determine the time of day and place where this type of activity would work best.
- Build slowly, perhaps just starting with a short walk around the yard, for example, before progressing to a walk around the block.
- Be aware of any discomfort or signs of overexertion. Talk to the person's doctor if this happens.
- Allow as much independence as possible, even if it means a lessthan-perfect garden or a scoreless tennis match.
- Investigate what kinds of exercise programs are available in your area. Senior centers may have group programs for people who enjoy exercising with others. Local malls often have walking clubs and provide a place to exercise when the weather is bad.
- Encourage physical activities. Spend time outside when the weather permits. Exercise often helps everyone sleep better.

### **Tailored Activity Program**

Participation in meaningful activity is known to promote well-being in older adults, and its potential to reduce symptoms of dementia is now receiving more attention. The Tailored Activity Program (TAP) is a home-based therapeutic plan. It is based on the environmental vulnerability/reduced stress threshold model, which asserts that with disease progression, dementia patients become increasingly vulnerable to their environment and experience lower thresholds for tolerating stimuli, which can result in behavioral disturbances. The intervention addresses this vulnerability by matching activities to cognitive and functional capabilities, as well as the previous roles, habits, and interests of the patient.

A preliminary study found that the TAP program improved overall levels of pleasure and engagement in the demented individuals, as assessed by their caregivers. It also reduced the extent to which caregivers were upset by the behavioral symptoms of the individuals they were caring for, and improved their sense of skill and personal control in dealing with behavioral problems.

# **Safety Recommendations**

People with AD become increasingly unable to take care of themselves. However, individuals will move through the disease in their own unique manner. Caregivers face the ongoing challenge of adapting to each change in the person's behavior and functioning. The following general principles may be helpful.

- Think prevention. It is very difficult to predict what a person with AD might do. Just because something has not yet occurred, does not mean it should not be cause for concern. Even with the bestlaid plans, accidents can happen. Therefore, checking the safety of the home will help control some of the potential problems that may create hazardous situations.
- 2. Adapt the environment. It is more effective to change the environment than to change most behaviors. While some AD behaviors can be managed with special medications prescribed by a doctor, many cannot. Changes in an environment must be made to decrease the hazards and stressors that accompany these behavioral and functional changes.
- 3. **Minimize danger**. By minimizing danger, you can maximize independence. A safe environment can be a less restrictive environment where the person with AD can experience increased security and more mobility.

#### Supervision

This issue needs careful evaluation and is certainly a safety concern. The following points should be considered.

Does the person with AD:

- become confused or unpredictable under stress?
- recognize a dangerous situation; for example, fire?
- know how to use the telephone in an emergency?
- know how to get help?
- stay content within the home?
- wander and become disoriented?
- show signs of agitation, depression, or withdrawal when left alone for any period of time?
- attempt to pursue former interests or hobbies that might now warrant supervision such as cooking, appliance repair, or woodworking?

# **Home Safety**

Prevention begins with a safety check of every room in the home. Use the following room-by-room checklist to identify potential hazards and to record any changes that need to be made. Products or gadgets necessary to improve home safety can be purchased at stores carrying hardware, electronics, medical supplies, and children's items.

Caregivers can make adaptations that modify and simplify without severely disrupting the home. A safe home can be a less stressful home for the person with AD, the caregiver, and family members.

# **Throughout the Home**

- Display emergency numbers and your home address near all telephones.
- Use a telephone answering machine when you cannot answer calls. The person with AD often is unable to take messages or may be a target for telephone exploitation by solicitors. When the answering machine is on, turn down the phone bell to avoid disruptive ringing.
- Install smoke alarms near all bedrooms; check their functioning and batteries frequently.
- Avoid the use of flammable and volatile compounds near gas water heaters. Do not store these materials in an area where a gas pilot light is used.
- Install secure locks on all outside doors and windows.
- Hide a spare house key outside in case the person with AD locks you out of the house.
- Avoid the use of extension cords if possible by placing lamps and appliances close to electrical outlets. Tack extension cords to the baseboards of a room to avoid tripping.
- Cover unused outlets with childproof plugs.
- Place red tape around floor vents, radiators, and other heating devices to deter the person with AD from standing on or touching a hot grid.
- Check all rooms for adequate lighting.
- Place light switches at the top and the bottom of stairs.
- Stairways should have at least one handrail that extends beyond the first and last steps. If possible, stairways should be carpeted or have safety grip strips.

- Keep all medications (prescription and over-the-counter) locked. Each bottle of prescription medicine should be clearly labeled with the patient's name, name of the drug, drug strength, dosage frequency, and expiration date. Child-resistant caps are available if needed.
- Keep all alcohol in a locked cabinet or out of reach of the person with AD. Drinking alcohol can increase confusion.
- If smoking is permitted at all, monitor while the person with AD is smoking. Remove matches, cigarettes, and ashtrays. With these reminders out of sight, the person may forget the desire to smoke.
- Avoid clutter, which can create confusion and danger. Throw out/recycle newspapers and magazines regularly. Keep all walk areas free of furniture.
- Keep plastic bags out of reach. A person with AD may choke or suffocate.
- Remove all guns or other weapons from the home, or safety proof them by installing safety locks or by removing ammunition and firing pins.
- Lock all power tools and machinery in the garage, workroom, or basement.
- Remove all poisonous plants from the home. Check with local nurseries or poison control centers for a list of poisonous plants.
- Keep fish tanks out of reach. The combination of glass, water, electrical pumps, and potentially poisonous aquatic life could be harmful to a curious person with AD.

### **Outside Approaches to the House**

- Keep steps sturdy and textured to prevent falls in wet or icy weather.
- Mark the edges of steps with bright or reflective tape.
- Consider a ramp with handrails into the home rather than steps.
- Eliminate uneven surfaces or walkways, hoses, or other objects that may cause a person to trip.
- Restrict access to a swimming pool by fencing it off with a locked gate, covering it, and keeping it closely supervised when in use.
  - In the patio area, remove the fuel source and fire starters from any grills when not in use, and supervise use when the person with AD is present.

#### Alzheimer's Disease

- Place a small bench or table by the entry door to hold parcels while unlocking the door.
- Make sure outside lighting is adequate. Light sensors that turn
  on lights automatically as you approach the house are available
  and may be useful. They also may be used in other parts of the
  home.
- Prune bushes and foliage well away from walkways and doorways.
- Consider a NO SOLICITING sign for the front gate or door.

# **Entryway**

- Remove scatter rugs and throw rugs.
- Use textured strips or nonskid wax on hardwood floors to prevent slipping.

#### Kitchen

- Install childproof door latches on storage cabinets and drawers designated for breakable or dangerous items. Lock away all household cleaning products, matches, knives, scissors, blades, small appliances, and valued china.
- If prescription or nonprescription drugs are kept in the kitchen, store them in a locked cabinet.
- Remove scatter rugs and foam pads from the floor.
- Remove knobs from the stove, or install an automatic shut-off switch.
- Do not use or store flammable liquids in the kitchen. Lock them in the garage or in an outside storage unit.
- Keep a night-light in the kitchen.
- Remove or secure the family "junk drawer." A person with AD may eat small items such as matches, hardware, erasers, plastics, etc.
- Remove artificial fruits and vegetables or food-shaped kitchen magnets, which might appear to be edible.
- Insert a drain trap in the kitchen sink to catch anything that may otherwise become lost or clog the plumbing.
- Consider dismantling the garbage disposal. People with AD may place objects or their own hands in the disposal.

#### **Bedroom**

- Use a night-light.
- Use an intercom device (often used for infants) to alert you to any noises indicating falls or a need for help. This also is an effective device for bathrooms.
- Remove scatter rugs.
- Remove portable space heaters. If you use portable fans, be sure that objects cannot be placed in the blades.
- Be cautious when using electric mattress pads, electric blankets, electric sheets, and heating pads, all of which may cause burns. Keep controls out of reach.
- Move the bed against the wall for increased security, or place the mattress on the floor.

#### **Bathroom**

- Do not leave a severely impaired person with AD alone in the bathroom.
- Remove the lock from the bathroom door to prevent the person with AD from getting locked inside.
- Place nonskid adhesive strips, decals, or mats in the tub and shower. If the bathroom is uncarpeted, consider placing these strips next to the tub, toilet, and sink.
- Use washable wall-to-wall bathroom carpeting to prevent slipping on wet tile floors.
- Use an extended toilet seat with handrails, or install grab bars beside the toilet.
- Install grab bars in the tub/shower. A grab bar in contrasting color to the wall is easier to see.
- Use a foam rubber faucet cover (often used for small children) in the tub to prevent serious injury should the person with AD fall.
- Use plastic shower stools and a hand-held showerhead to make bathing easier.
- In the shower, tub, and sink, use a single faucet that mixes hot and cold water to avoid burns.
- Adjust the water heater to 120 degrees to avoid scalding tap water.

- Insert drain traps in sinks to catch small items that may be lost or flushed down the drain.
- Store medications (prescription and nonprescription) in a locked cabinet. Check medication dates and throw away outdated medications.
- Remove cleaning products from under the sink, or lock them away.
- Use a night-light.
- Remove small electrical appliances from the bathroom. Cover electrical outlets. If men use electric razors, have them use a mirror outside the bathroom to avoid water contact.

# **Living Room**

- Clear all walk areas of electrical cords.
- Remove scatter rugs or throw rugs. Repair or replace torn carpet.
- Place decals at eye level on sliding glass doors, picture windows, or furniture with large glass panels to identify the glass pane.
- Do not leave the person with AD alone with an open fire in the fireplace, or consider alternative heating sources. Remove matches and cigarette lighters.
- Keep the controls for cable or satellite TV, VCR, and stereo system out of sight.

#### **Laundry Room**

- Keep the door to the laundry room locked if possible.
- Lock all laundry products in a cabinet.
- Remove large knobs from the washer and dryer if the person with AD tampers with machinery.
- Close and latch the doors and lids to the washer and dryer to prevent objects from being placed in the machines.

#### **Behavior Management**

Although a number of behavior and sensory problems may accompany Alzheimer's disease, not every person will experience the disease in exactly the same way. As the disease progresses, particular behavioral changes can create safety problems. The person with AD may or may not have these symptoms. However, should these behaviors occur, the following safety recommendations may help reduce risks.

## Wandering

- Remove clutter and clear the pathways from room to room to allow the person with AD to move about more freely.
- Make sure floors provide good traction for walking or pacing.
   Use nonskid floor wax or leave floors unpolished. Secure all rug edges, eliminate throw rugs, or install nonskid strips. The person with AD should wear nonskid shoes or sneakers.
- Place locks on exit doors high or low on the door out of direct sight. Consider double locks that require a key. Keep a key for yourself and hide one near the door for emergency exit purposes.
- Use loosely fitting doorknob covers so that the cover turns instead of the actual knob. (Due to the potential hazard they could cause if an emergency exit is needed, locked doors and doorknob covers should be used only when a caregiver is present.)
- Install safety devices found in hardware stores to limit the distance that windows can be opened.
- If possible, secure the yard with fencing and a locked gate. Use door alarms such as loose bells above the door or devices that ring when the doorknob is touched or the door is opened.
- Divert the attention of the person with AD away from using the door by placing small scenic posters on the door; placing removable gates, curtains, or brightly colored streamers across the door; or wallpapering the door to match any adjoining walls.
- Place STOP, DO NOT ENTER, or CLOSED signs in strategic areas on doors.
- Reduce clues that symbolize departure such as shoes, keys, suitcases, coats, or hats.
- Obtain a medical identification bracelet for the person with AD with the words "memory loss" inscribed along with an emergency telephone number. Place the bracelet on the person's dominant hand to limit the possibility of removal, or solder the bracelet closed.
- Place labels in garments to aid in identification. Check with the local Alzheimer's Association about the Safe Return program.
- Keep an article of the person's worn, unwashed clothing in a plastic bag to aid in finding someone with the use of dogs.

- Notify neighbors of the person's potential to wander or become lost. Alert them to contact you or the police immediately if the individual is seen alone and on the move.
- Give local police, neighbors, and relatives a recent picture, along with the name and pertinent information about the person with AD, as a precaution should he or she become lost. Keep extra pictures on hand.
- Consider making an up-to-date home video of the person with AD.
- Do not leave a person with AD who has a history of wandering unattended.

## **Rummaging/Hiding Things**

- Lock up all dangerous or toxic products, or place them out of the person's reach.
- Remove all old or spoiled food from the refrigerator and cupboards. A person with AD may rummage for snacks but may lack the judgment or taste to rule out spoiled foods.
- Simplify the environment by removing clutter or valuable items that could be misplaced, lost, or hidden by the person with AD. These include important papers, checkbooks, charge cards, and jewelry.
- If your yard has a fence with a locked gate, place the mailbox outside the gate. People with AD often hide, lose, or throw away mail. If this is a serious problem, consider obtaining a post office box.
- Create a special place for the person with AD to rummage freely or sort (for example, a chest of drawers, a bag of selected objects, or a basket of clothing to fold or unfold). Often, safety problems occur when the person with AD becomes bored or does not know what to do.
- Provide the person with AD a safe box, treasure chest, or cupboard to store special objects.
- Close access to unused rooms, thereby limiting the opportunity for rummaging and hiding things.
- Search the house periodically to discover hiding places. Once found, these hiding places can be discreetly and frequently checked.

- Keep all trashcans covered or out of sight. The person with AD may not remember the purpose of the container or may rummage through it.
- Check trash containers them before emptying them in case something has been hidden there or accidentally thrown away.

## Hallucinations, Illusions, and Delusions

Due to the complex changes occurring in their brain, people with AD may see or hear things that have no basis in reality. Hallucinations come from within the brain and involve hearing, seeing, or feeling things that are not really there. For example, a person with AD may see children playing in the living room when no children exist. Illusions differ from hallucinations because the person with AD is misinterpreting something that actually does exist. Shadows on the wall may look like people, for example. Delusions are persistent thoughts that the person with AD believes are true but in reality, are not. Often, stealing is suspected, for example, but cannot be verified.

It is important to seek medical evaluation if a person with AD has ongoing disturbing hallucinations, illusions, or delusions. Often, these symptoms can be treated with medication or behavior management techniques. With all of the above symptoms, the following environmental adaptations also may be helpful.

- Paint walls a light color to reflect more light. Use solid colors, which are less confusing to an impaired person than a patterned wall. Large, bold prints (for example, florals in wallpaper or drapes) may cause confusing illusions.
- Make sure there is adequate lighting, and keep extra bulbs handy in a secured place. Dimly lit areas may produce confusing shadows or difficulty with interpreting everyday objects.
- Reduce glare by using soft light or frosted bulbs, partially closing blinds or curtains, and maintaining adequate globes or shades on light fixtures.
- Remove or cover mirrors if they cause the person with AD to become confused or frightened.
- Ask if the person can point to a specific area that is producing confusion. Perhaps one particular aspect of the environment is being misinterpreted.
- Vary the home environment as little as possible to minimize the potential for visual confusion. Keep furniture in the same place.
- Have the person with AD avoid watching violent or disturbing television programs. The person with AD may believe the story is real.

Do not confront the person with AD who becomes aggressive.
 Withdraw and make sure you have access to an exit as needed.

## **Sensory Impairment**

Alzheimer's disease can cause changes in a person's ability to interpret what they see, hear, taste, feel, or smell, even though the sense organs may still be intact. The person with AD should be evaluated periodically by a physician for any such changes that may be correctable with glasses, dentures, hearing aids, or other treatments.

### Vision

People with AD may experience a number of changes in visual abilities. For example, they may lose their ability to comprehend visual images. Although there is nothing physically wrong with their eyes, people with AD may no longer be able to interpret accurately what they see due to changes in their brain. Also, their sense of perception and depth may be altered. These changes can cause safety concerns.

- Create color contrast between floors and walls to help the person see depth. Floor coverings are less visually confusing if they are a solid color.
- Use dishes and placemats in contrasting colors for easier identification.
- Mark the edges of steps with brightly colored strips of tape to outline changes in elevation.
- Place brightly colored signs or simple pictures on important rooms (the bathroom, for example) for easier identification.
- Be aware that a small pet that blends in with the floor or lies in walkways may be a hazard. The person with AD may trip over a small pet.

### Smell

A loss or decrease in smell often accompanies Alzheimer's disease.

- Install good quality smoke detectors and check them frequently.
   The person with AD may not smell smoke or may not associate it with danger.
- Keep refrigerators clear of spoiled foods.

### Touch

People with AD may experience loss of sensation or may no longer be able to interpret feelings of heat, cold, or discomfort.

- Adjust water heaters to 120 degrees to avoid scalding tap water.
   Most hot water heaters are set at 150 degrees, which can cause burns.
- Color code separate water faucet handles, with red for hot and blue for cold.
- Place a sign on the oven, coffee maker, toaster, crock-pot, iron, or other potentially hot appliances that says DO NOT TOUCH or STOP! VERY HOT. The person with AD should not use appliances without supervision. Unplug appliances when not in use.
- Use a thermometer to tell you whether the water in the bathtub is too hot or too cold.
- Remove furniture or other objects with sharp corners or pad them to reduce potential for injury.

### Taste

People with AD may lose taste sensitivity. As their judgment declines, they also may place dangerous or inappropriate things in their mouth.

- If possible, keep a spare set of dentures. If the person keeps removing dentures, check for correct fit.
- Keep all condiments such as salt, sugar, or spices away from easy access if you see the person with AD using excess amounts. Too much salt, sugar, or spice can be irritating to the stomach or cause other health problems.
- Remove or lock up medicine cabinet items such as toothpaste, perfume, lotions, shampoos, rubbing alcohol, or soap, which may look and smell like edible items to the person with AD.
- Consider a childproof latch on the refrigerator, if necessary.
- Keep the poison control number by the telephone. Keep a bottle of Ipecac (vomit inducing) available but use only with instructions from poison control or 911.
- Keep pet litter boxes inaccessible to the person with AD. Do not store pet food in the refrigerator.
- Learn the Heimlich maneuver or other techniques to use in case of choking. Check with your local Red Cross for more information and instruction.

## Hearing

People with AD may have normal hearing, but they may lose their ability to interpret what they hear accurately. This may result in confusion or over-stimulation.

- Avoid excessive noise in the home such as having the stereo and the TV on at the same time.
- Be sensitive to the amount of noise going on outside, and close windows or doors, if necessary.
- Avoid large gatherings of people in the home if the person with AD shows signs of agitation or distress in crowds.
- Check hearing aid batteries and functioning frequently.

## **Driving**

Driving is a complex activity that demands quick reactions, alert senses, and split-second decision-making. For a person with AD, driving becomes increasingly more difficult. Memory loss, impaired judgment, disorientation, impaired visual and spatial perception, slow reaction time, diminished attention span, inability to recognize cues such as stop signs and traffic lights can make driving particularly hazardous.

People with AD who continue to drive can be a danger to themselves, their passengers, and the community at large. As the disease progresses, they lose driving skills and must stop driving. Unfortunately, people with AD often cannot recognize when they should no longer drive. This is a tremendous safety concern. It is extremely important to have the impaired person's driving abilities carefully evaluated.

Often, it is the caregiver, a family member, neighbor, or friend who becomes aware of the safety hazards. If a person with AD experiences one of more of the following problems, it may be time to limit or stop driving.

Does the person with AD:

- get lost while driving in a familiar location?
- fail to observe traffic signals?
- drive at an inappropriate speed?
- become angry, frustrated, or confused while driving?
- make slow or poor decisions?

Explaining to the person with AD that he or she can no longer drive can be extremely difficult. Loss of driving privileges may represent a tremendous loss of independence, freedom, and identity. It is a significant concern for the person with AD and the caregiver. The issue of not driving may produce anger, denial, and grief in the person with AD, as well as guilt and anxiety in the caregiver. Family and concerned professionals need to be both sensitive and firm. Above all, they should be persistent and consistent. The doctor of a person with AD can assist the family with the task of restricting driving. Talk with the doctor about your concerns. Most people will listen to their doctor. Ask the doctor to advise the person with AD to reduce his or her driving, go for a driving evaluation or test, or

stop driving altogether. An increasing number of States have laws requiring physicians to report AD and related disorders to the Department of Motor Vehicles. The Department of Motor Vehicles then is responsible for retesting the at-risk driver. Testing should occur regularly, at least yearly.

When dementia impairs driving and the person with AD continues to insist on driving, a number of different approaches may be necessary. Work as a team with family, friends, and professionals and use a single, simple explanation for the loss of driving ability such as: "You have a memory problem, and it is no longer safe to drive." "You cannot drive because you are on medication." or "The doctor has prescribed that you no longer drive."

- Have the doctor write on a prescription pad DO NOT DRIVE.
   Ask the doctor to write to the Department of Motor Vehicles or Department of Public Safety saying this person should no longer drive. Show the letter to the person with AD as evidence.
- Offer to drive.
- Walk when possible, and make these outings special events.
- Use public transportation or any special transportation provided by community organizations. Ask about senior discounts or transportation coupons. The person with AD should not take public transportation unsupervised.
- Park the car at a friend's home.
- Hide the car keys.
- Exchange car keys with a set of unusable keys. Some people with AD are in the habit of carrying keys.
- Place a large note under the car hood requesting that any mechanic call you before doing work requested by the person with AD.
- Have a mechanic install a "kill switch" or alarm system that disengages the fuel line to prevent the car from starting.
- Consider selling the car and putting aside the money saved from insurance, repairs, and gasoline for taxi funds.
- Do not leave a person with AD alone in a parked car.

## **Natural Disaster Safety**

Natural disasters come in many forms and degrees of severity. They seldom give warning, and they call upon good judgment and ability to follow through with crisis plans. People with AD are at a serious disadvantage. Their impairments in memory and reasoning severely limit their ability to act appropriately in crises.

It is always important to have a plan of action in case of fire, earthquake, flood, tornado, or other disasters. Specific home safety precautions may apply and environmental changes may be needed. The American Red Cross is an excellent resource for general safety information and preparedness guides for comprehensive planning. If there is a person with AD in the home, the following precautions apply:

- Get to know your neighbors, and identify specific individuals who would be willing to help in a crisis. Formulate a plan of action with them should the person with AD be unattended during a crisis.
- Give neighbors a list of emergency numbers of caregivers, family members, and primary medical resources.
- Educate neighbors beforehand about the person's specific disabilities, including inability to follow complex instructions, memory loss, impaired judgment, and probable disorientation and confusion. Give examples of some of the simple one-step instructions that the person may be able to follow.
- Have regular emergency drills so that each member of the household has a specific task. Realize that the person with AD cannot be expected to hold any responsibility in the crisis plan and that someone will need to take primary responsibility for supervising the individual.
- Always have at least an extra week's supply of any medical or personal hygiene items critical to the person's welfare, such as:
  - food and water
  - medications
  - incontinence undergarments
  - hearing aid batteries
  - glasses
- Be sure that the person with AD wears an identification bracelet stating "memory loss" should he or she become lost or disoriented during the crisis. Contact your local Alzheimer's Association chapter and enroll the person in the Safe Return program.
- Under no circumstances should a person with AD be left alone following a natural disaster. Do not count on the individual to stay in one place while you go to get help. Provide plenty of reassurance.

## The Stigma of Alzheimer's Disease

Stigma is the use of negative labels to identify a person living with a disability or illness. When someone appears to be different than us, we may view him or her in a negative stereotyped manner. People who have identities that society values negatively are said to be stigmatized. Concern over stigma and denial can delay diagnosis of Alzheimer's disease for years, meaning patients do not receive treatment that could slow its progress. When spouses or other relatives who care for patients are concerned about the stigma associated with Alzheimer's, delay of the diagnosis averages 6 years after symptoms first appear.

While facing Alzheimer's disease is never easy, getting a diagnosis is an essential step to managing and treating the disease. Living with this in silence can isolate people with Alzheimer's disease and their caregivers, leaving them without critical support, resources, and proper treatment.

Lack of knowledge about the disease is a barrier to diagnosis for two in five caregivers (40 percent), suggesting that more education is needed. While the majority (92 percent) of caregivers report that they are aware of medications that may slow the progression of symptoms, only half (51 percent) of caregivers are aware of the opportunity for combination therapy. Remarkably, many caregivers who mentioned fear of stigma and/or denial as a reason for delay in diagnosis appear to have largely overcome their fear and are more likely than those who did not struggle with stigma and denial to say they are extremely or very knowledgeable about Alzheimer's disease (72 percent vs. 59 percent).

### Research on AD Prevention

## **Physical Activity**

Accumulating evidence suggests that physical activity may be good for our brains as well as our hearts, waistlines, and ability to carry out activities of daily living. Epidemiologic studies have found associations between physical activity and improved cognitive skills or reduced AD risk. For example, investigators looked at the relationship of physical activity and AD risk in about 1,700 adults aged 65 years and older over a 6-year period. They found that the risk of AD was 35 to 40 percent lower in those who exercised for at least 15 minutes 3 or more times a week than in those who exercised fewer than 3 times a week.

Scientists have sought to confirm these associations in animal studies, hoping to clarify why physical activity might be related to reduced risk of cognitive decline and AD. For example, studies in older rats and mice have found that exercise increases the number of small blood vessels that supply blood to the brain and increases the number of connections between nerve cells. Other research has

shown exercise to raise the level of specific brain-growth factors in an area of the brain that is particularly important to memory and learning.

Both epidemiologic and animal studies point to associations and help to explain them. However, epidemiologic studies can't tell us whether a true cause-and-effect relationship exists between a particular factor and AD risk. For example, people who exercise tend to be healthier in other ways, such as having decreased rates of heart disease or diabetes. They may also have healthier lifestyles, such as eating a nutritious diet. This means that even if people who exercise are less likely to develop AD, we don't know whether this is due to the exercise or the more healthful eating or other lifestyle differences that distinguish them from inactive people.

One completed trial used functional magnetic resonance imaging (MRI) tests to measure changes in brain activity in older adults before and after a 6-month program of brisk walking. Results showed that brain activity increased in specific brain regions as the participants' cardiovascular fitness increased. A similar study showed that brain volume increased as a result of a walking program.

These findings strongly suggest a biological basis for the role of aerobic fitness in helping to maintain the health and cognitive functioning of adults as they age, at least in the short term.

## **Dietary Factors**

A number of studies suggest that how we eat may be linked to our risk of developing AD. This is another important area of current AD research. A nutritious diet—a diet that includes lots of fruits, vegetables, and whole grains and is low in fat and added sugar—can reduce the risk of many chronic diseases, including heart disease, type 2 diabetes, and obesity. Animal studies, epidemiologic studies, and clinical trials are looking at whether a healthy diet also can help preserve cognitive function or even reduce AD risk.

Studies have examined foods that are rich in antioxidants and anti-inflammatory components to find out whether those foods affect age-related changes in the brain. One study found that curcumin, the main ingredient of turmeric (a spice used in curry), can suppress the build-up of harmful beta-amyloid in the brains of rodents. Another study, in AD transgenic mice (those that are specially bred to have features of AD), found that DHA (docosahexaenoic acid, a type of omega-3 fatty acid found in some fish) reduced the presence of beta-amyloid and plaques. Other research has shown that older dogs perform better on learning tasks when they eat a diet rich in antioxidants and live in an "enriched" environment with many opportunities to play and interact with others.

In addition, studies in rats and mice have shown that dietary supplementation with blueberries, strawberries, and cranberries can improve cognitive function,

both during normal aging and in animals that have been bred to develop AD. Scientists are beginning to identify some of the chemicals responsible for these berries' beneficial effects and think that the chemicals may act by neutralizing free radicals. This may reduce inflammation or stimulate neurons to protect themselves better against some of the adversities of aging and AD.

Several epidemiologic studies have shown an association between eating a diet rich in vegetables (especially green leafy vegetables and cruciferous vegetables like broccoli) and a reduced rate of cognitive decline. Researchers speculate that the beneficial effect may come from the antioxidant and folate content of the vegetables.

These results are interesting, but in their normal daily lives, people typically consume many different foods and nutrients. With this in mind, some investigators have conducted epidemiologic studies to examine a group's entire dietary pattern. One of these studies showed a reduced risk of AD in those who ate the "Mediterranean diet"—a diet that includes many fruits, vegetables, and beans; moderate amounts of fish; low-to-moderate amounts of dairy foods; small amounts of meat and poultry; regular but moderate amounts of wine; and olive oil.

## **Social & Cognitive Activities**

Observations of nursing home residents and older people living in the community have suggested a link between social engagement and cognitive abilities. Having many friends and acquaintances and participating in many social activities also is associated with reduced cognitive decline and decreased risk of dementia in older adults. For example, the Chicago Health and Aging Project showed that more social networks and a higher level of social engagement were associated with a higher level of cognitive function at the beginning of the study. These factors also were related to a reduced rate of cognitive decline over time. Studies have also shown that keeping the brain active is associated with reduced AD risk. In the Religious Orders Study, for example, investigators periodically asked more than 700 participants—older nuns, priests, and religious brothers—to describe the amount of time they spent in seven information-processing activities. These activities included listening to the radio, reading newspapers, playing puzzle games, and going to museums. After following the participants for 4 years, the investigators found that the risk of developing AD was 47 percent lower, on average, for those who did the activities most often than for those who did them least frequently.

Other studies have shown similar results. In addition, a growing body of research suggests that, even in the presence of AD plaques, the more formal education a person has, the better his or her memory and learning abilities.

Another study supports the value of lifelong learning and mentally stimulating activity. It showed that during early and middle adulthood, cognitively healthy

older people had engaged in more mentally stimulating activities and spent more hours doing them than did those who ultimately developed AD. Other studies have shown that people who are bilingual or multilingual seem to develop AD at a later age than do people who only speak one language.

The reasons for this apparent link between social engagement or intellectual stimulation and AD risk aren't entirely clear, but scientists suggest four possibilities:

- Such activities may protect the brain in some way, perhaps by establishing "cognitive reserve." (Cognitive reserve is the brain's ability to operate effectively even when some function is disrupted or the amount of damage that the brain can sustain before changes in cognition are evident.)
- These activities may help the brain become more adaptable and flexible in some areas of mental function so that it can compensate for declines in other areas.
- People who engage in these activities may have other lifestyle factors that protect them against developing AD.
- Less engagement with other people or in intellectually stimulating activities could be the result of very early effects of the disease rather than its cause.

The only way to really evaluate some of these possibilities is to test them in a controlled way in clinical trials. Several clinical trials have examined whether memory training and similar types of mental skills training can actually improve the cognitive abilities of healthy older adults and people with mild AD. In the Advanced Cognitive Training for Independent and Vital Elderly (ACTIVE) trial, for example, certified trainers provided 10 sessions of memory training, reasoning training, or processing-speed training to healthy adults 65 years old and older. The sessions improved participants' mental skills in the area in which they were trained. Even better, these improvements persisted for up to 5 years after the training was completed.

## **Supplemental Information**

### Primary Prevention of Alzheimer's Disease

Ettore Bergamini and Gabriella Cavallini (2019). Primary Prevention of Alzheimer's Disease (AD), Geriatric Medicine and Gerontology, Edward T. Zawada Jr., IntechOpen, DOI: 10.5772/intechopen.85418. https://www.intechopen.com/books/geriatric-medicine-and-gerontology/primary-prevention-of-alzheimer-s-disease-ad- Published under CC BY 3.0 license

# Non-pharmacological Treatment of Alzheimer's, Redirecting Alzheimer Strategy - Tracing Memory Loss to Self-Pathology, Denis Larrivee,

Terezia Fertalova and Iveta Ondriova (2019). Non-pharmacological Treatment of Alzheimer's, Redirecting Alzheimer Strategy - Tracing Memory Loss to Self-Pathology, Denis Larrivee, IntechOpen, DOI: 10.5772/intechopen.84893. Available from: https://www.intechopen.com/books/redirecting-alzheimer-strategy-tracing-memory-loss-to-self-pathology/non-pharmacological-treatment-of-alzheimer-s Published under CC BY 3.0 license

# Introductory Chapter: Alzheimer's Disease—The Most Common Cause of Dementia, Advances in Dementia Research

Md. Sahab Uddin and Ghulam Md. Ashraf (2018). Introductory Chapter: Alzheimer's Disease—The Most Common Cause of Dementia, Advances in Dementia Research, Ghulam Md. Ashraf, IntechOpen, DOI: 10.5772/intechopen.82196. Available from: https://www.intechopen.com/books/advances-in-dementia-research/introductory-chapter-alzheimer-s-disease-the-most-common-cause-of-dementia

### Epidemiology of Alzheimer's disease

Xu, W., Ferrari, C., & Wang, H. X. (2013). Epidemiology of Alzheimer's disease. In I. Zerr (Ed.), *Understanding Alzheimer's Disease* (pp. 329-358). InTech. Published under CC BY 3.0 license.

### Potential therapeutic strategies to prevent the progression of Alzheimer to disease states

Aso, E., & Ferrer, I. (2013). Potential therapeutic strategies to prevent the progression of Alzheimer to disease states. In I. Zerr (Ed.), *Understanding Alzheimer's Disease* (pp. 251-302). InTech. Published under CC BY 3.0 license

### Animal assisted therapy and activities in Alzheimer's disease

Cevizci, S., Sen, H. M., Günes, F., & Karaahmet, E. (2013). Animal assisted therapy and activities in Alzheimer's disease. In I. Zerr (Ed.), *Understanding Alzheimer's Disease* (pp. 303-325). InTech. Published under CC BY 3.0 license

### Prevention of Alzheimer's disease: Intervention studies

Mangialasche, F., Xu, W., & Kivipelto, M. (2013). Prevention of Alzheimer's disease: Intervention studies. In I. Zerr (Ed.), *Understanding Alzheimer's Disease* (pp. 452-484). InTech. Published under CC BY 3.0 license New frontiers in Alzheimer's disease diagnosis

Llorens, F., Nuhn, S., Peter, C., Zerr, I., & Stoeck, K. (2015). New frontiers in Alzheimer's disease diagnosis. In I. Zerr (Ed.), *Alzheimer's Disease - Challenges for the Future* (pp. 257-294). InTech. Published under CC BY 3.0 license

### Alzheimer's disease: A clinical perspective

Xu, W., Marseglia, A., Ferrari, C., & Wang, H. X. (2013). Alzheimer's disease: A clinical perspective. In U. Kishore (Ed.), *Neurodegenerative Diseases* (pp. 3-33). InTech. Published under CC BY 3.0 license Vascular dementia and Alzheimer's disease: Is there a difference?

Ramdane, S. (2012). Vascular dementia and Alzheimer's disease: Is there a difference? In L. M. Martins & S. H.Y. Loh (Eds.), *Neurodegeneration* (pp. 231-256). InTech. Published under CC BY 3.0 license <u>Late-Onset Alzheimer's Disease: Risk Factors, Clinical Diagnosis and the Search for Biomarkers</u> Herrera-Rivero, M. (2013). Late-Onset Alzheimer's Disease: Risk Factors, Clinical Diagnosis and the Search for Biomarkers. In U. Kishore (Ed.), *Neurodegenerative Diseases*. InTech. Published under CC BY 3.0 license

### Alzheimer's Disease Clinical and Research Update for Health Care Practitioners

DeFina, P. A., Moser, R. S., Glenn, M., Lichtenstein, J. D., & Fellus, J. (2013). Alzheimer's disease clinical and research update for health care practitioners. *Journal of aging research*, 2013. Published under CC BY 3.0 license

### Resources

### **Alzheimer's Association**

225 North Michigan Ave.

**Suite 1700** 

Chicago, IL 60601

Website: www.alz.org

### **Alzheimer's Disease Cooperative Study**

University of California, San Diego 9500 Gilman Drive - 0949

La Jolla, CA 92093-0949

858-622-5880

Website: http://antimony.ucsd.edu/

## Alzheimer's Disease Education and Referral (ADEAR) Center

PO Box 8250

Silver Spring, MD 20907

1-800-438-4380

Website: www.alzheimers.org

### **Children of Aging Parents**

1609 Woodbourne Road, Suite 302A

Levittown, PA 19057-1511

1-800-227-7294

Website: www.caps4caregivers.org

### **Eldercare Locator**

1-800-677-1116

Web site: www.eldercare.gov

### **Family Caregiving Alliance**

690 Market Street, Suite 600

San Francisco, CA 94104

415-434-3388

### **National Institute on Aging Information Center**

PO Box 8057

Gaithersburg, MD 20898-8057

1-800-222-2225

1-800-222-4225 (TTY)

Website: www.nia.nih.gov

### **Partnership for Caring**

1620 Eye Street NW, Suite 202

Washington, DC 20006

1-800-989-9455

Website: www.partnershipforcaring.org

### **Well Spouse Foundation**

63 West Main Street, Suite H

Freehold, NJ 07728

1-800-838-0879

Website: www.wellspouse.org

### References

Albert, M. S., DeKosky, S. T., Dickson, D., Dubois, B., Feldman, H. H., Fox, N. C., ... & Snyder, P. J. (2011). The diagnosis of mild cognitive impairment due to Alzheimer's disease: Recommendations from the National Institute on Aging-Alzheimer's Association workgroups on diagnostic guidelines for Alzheimer's disease. Alzheimer's & dementia, 7(3), 270-279.

Allan, C. L., Behrman, S., & Ebmeier, K. P. (2013). Early diagnosis beneficial in Alzheimer's disease. The Practitioner, 257(1757), 15-19.

Aso, E., & Ferrer, I. (2013). Potential therapeutic strategies to prevent the progression of Alzheimer to disease states. In I. Zerr (Ed.), *Understanding Alzheimer's Disease* (pp. 251-302). InTech.

Aulston, B. D., Odero, G. L., Aboud, Z., & Glazner, G. W. (2013). Alzheimer's disease and diabetes. In I. Zerr (Ed.), *Understanding Alzheimer's Disease* (pp. 410-449). InTech.

Aysegul Uludag, Sibel Cevizci and Ahmet Uludag (2015). Epidemiology of Alzheimer's Disease with the Projection of Falls Among the Aged Population, Alzheimer's Disease - Challenges for the Future, Prof. Inga Zerr (Ed.), InTech

Benjamin, R. M. (2013). A plan to address Alzheimer's disease. Public Health Reports, 128(2), 76.

Bertram, L., Lill, C. M., & Tanzi, R. E. (2010). The genetics of Alzheimer disease: back to the future. Neuron, 68(2), 270-281.

Budson, A. E., & Solomon, P. R. (2012). New criteria for Alzheimer disease and mild cognitive impairment: implications for the practicing clinician. *The neurologist*, 18(6), 356-363.

Buss SS, Fried PJ & Pascual-Leone A. (2019). Therapeutic noninvasive brain stimulation in Alzheimer's disease and related dementias. Current Opinion in Neurology, 32, 292-304.

De Breucker Sandra, P. T., & Jean-Christophe, B. (2015). The Alzheimer's Patient in the Emergency Department—Specificities of Care.

de Heus RAA, Olde Rikkert MGM, Tully PJ, Lawlor BA, Claassen JAHR & NILVAD Study Group. (2019). Blood Pressure Variability and Progression of Clinical Alzheimer Disease. Hypertension, 74, 1172-1180.

DeFina, P. A., Moser, R. S., Glenn, M., Lichtenstein, J. D., & Fellus, J. (2013). Alzheimer's disease clinical and research update for health care practitioners. *Journal of aging research*, 2013.

Herrera-Rivero, M., & Aranda-Abreu, E. (2011). Therapeutics of Alzheimer's disease. In R. C. Chang (Ed.), Advanced Understanding of Neurodegenerative Diseases (pp. 193-212). InTech.

Herrera-Rivero, M. (2013). Late-Onset Alzheimer's Disease: Risk Factors, Clinical Diagnosis and the Search for Biomarkers. In U. Kishore (Ed.), Neurodegenerative Diseases. In Tech

Herrup, K. (2010). Reimagining Alzheimer's disease—an age-based hypothesis. The Journal of Neuroscience, 30(50), 16755-16762.

Jack Jr, C. R., Albert, M., Knopman, D. S., McKhann, G. M., Sperling, R. A., Carillo, M., ... & Phelps, C. H. (2011). Introduction to revised criteria for the diagnosis of Alzheimer's disease: National Institute on Aging and the Alzheimer Association workgroups. *Alzheimer's & dementia: the journal of the Alzheimer's Association*, 7(3), 257.

Kalaria, R. N., Akinyemi, R., & Ihara, M. (2012). Does vascular pathology contribute to Alzheimer changes? Journal of the neurological sciences, 322(1), 141-147.

Kapoor, S. (2012). Alzheimer's Disease—Emerging New Potential Therapeutic Modalities. American journal of Alzheimer's disease and other dementias, 27(8), 563-563.

Liesz A. (2019). The vascular side of Alzheimer's disease. Science, 365, 223-224

Llorens, F., Nuhn, S., Peter, C., Zerr, I., & Stoeck, K. (2015). New frontiers in Alzheimer's disease diagnosis. In I. Zerr (Ed.), Alzheimer's Disease - Challenges for the Future (pp. 257-294). InTech.

Mangialasche, F., Xu, W., & Kivipelto, M. (2013). Prevention of Alzheimer's disease: Intervention studies. In I. Zerr (Ed.), *Understanding Alzheimer's Disease* (pp. 452-484). InTech.

McKhann, G. M., Knopman, D. S., Chertkow, H., Hyman, B. T., Jack, C. R., Kawas, C. H., ... & Mohs, R. C. (2011). The diagnosis of dementia due to Alzheimer's disease: Recommendations from the National Institute on Aging-Alzheimer's Association workgroups on diagnostic guidelines for Alzheimer's disease. *Alzheimer's & dementia*, 7(3), 263-269.

Proietti R, AlTurki A, Vio R, Licchelli L, Rivezzi F, Marafi M, et al. (2020). The association between atrial fibrillation and Alzheimer's disease: fact or fallacy? A systematic review and meta-analysis. Journal of Cardiovascular Medicine, 21, 106-112.

Ramdane, S. (2012). Vascular dementia and Alzheimer's disease: Is there a difference? In L. M. Martins & S. H.Y. Loh (Eds.), Neurodegeneration (pp. 231-256). InTech.

Renteria ME, Mitchell BL & de Lara AM. (2020). Genetic testing for Alzheimer's disease: trends, challenges and ethical considerations. Current Opinion in Psychiatry, 33, 136-140.

 $Selkoe\ DJ.\ (2019).\ Early\ network\ dysfunction\ in\ Alzheimer's\ disease.\ Science,\ 365,\ 540-541.$ 

Sperling, R. A., Aisen, P. S., Beckett, L. A., Bennett, D. A., Craft, S., Fagan, A. M., ... & Park, D. C. (2011). Toward defining the preclinical stages of Alzheimer's disease: Recommendations from the National Institute on Aging-Alzheimer's Association workgroups on diagnostic guidelines for Alzheimer's disease. *Alzheimer's & Dementia*, 7(3), 280-292.

Stukas, S., Kulic, I., Zareyan, S., & Wellington, C. L. (2015). Lipids and Lipoproteins in Alzheimer's Disease.

van Loenhoud AC, van der Flier WM, Wink AM, Dicks E, Groot C, Twisk J, et al. (2019). Cognitive reserve and clinical progression in Alzheimer disease: A paradoxical relationship. Neurology, 93, e334-e346.

Xu, W., Marseglia, A., Ferrari, C., & Wang, H. X. (2013). Alzheimer's disease: A clinical perspective. In U. Kishore (Ed.), Neurodegenerative Diseases (pp. 3-33). InTech.

Xu, W., Ferrari, C., & Wang, H. X. (2013). Epidemiology of Alzheimer's disease. In I. Zerr (Ed.), Understanding Alzheimer's Disease (pp. 329-358). InTech.

Zhu, C. W., Sano, M., Ferris, S. H., Whitehouse, P. J., Patterson, M. B., & Aisen, P. S. (2013). Health-Related Resource Use and Costs in Elderly Adults with and without Mild Cognitive Impairment. *Journal of the American Geriatrics Society*, 61(3), 396-402.

## Post-Test

1.	Individuals with Alzheimer's Disease live for an average of after they are diagnos A. 2-4 years B. 4-6 years C. 6-8 years D. 8-10 years	ed. (p. 3)
2.	healthy aging, most types of brain neurons are not lost in large numbers. (p. 4) . True B. False	
3.	What are the three most evident abnormalities found in the brains of people who have of Alzheimer's? (p. 5)  A. Amyloid plaques, neurofibrillary tangles, nerve cell death  B. APOE, BIN1, CLU  C. Homocysteine, tau, free radicals  D. Inflammation, oxidative damage, aluminum deposits	ied with
4.	ore than 90 percent of AD cases occur in people older than 60. (p. 6) . True B. False	
5.	The causes of late-onset Alzheimer's most likely include a combination of genetic, envir and lifestyle factors. (p. 7) A. True B. False	onmental
6.	Symptoms of Alzheimer's disease often become worse in the evening; this is known as "lunarization". (p. 10-11) A. True B. False	
7.	is one of the most common early signs of dementia. (p. 11-12)  A. Forgetting recently learned information  B. Forgetting the day of the week  C. Occasionally forgetting why you came into a room  D. Finding it challenging to balance a checkbook	
8.	In which stage of Alzheimer's Disease do others typically begin to notice deficiencies?  A. Stage 2  B. Stage 3  C. Stage 4  D. Stage 5	(p. 12-15)
9.	Currently, the only way to definitively diagnose Alzheimer's Disease is (p. 17 A. autopsy B. the Mini Mental State Examination C. neuroimaging D. APOE testing	1
10.	Individuals with Alzheimer's disease generally score 30 points or more on the Mini Meni Examination. (p. 18) A. True B. False	al State
11.	APOE testing can confirm whether someone will develop Alzheimer's disease. (p. 20) A. True B. False	

12.	A therapy patient who is taking Namenda for moderate symptoms of AD should be monitored carefully during gait activities because is a common side effect of the medication.  (p. 21-22) A. weakness B. bradykinesia C. tachycardia D. dizziness	
13.	Ginkgo biloba has been found to be effective in reducing the development of dementia in older people. (p. 23) A. True B. False	
14.	The vast majority of individuals living with Alzheimer's disease are cared for at home by family members. (p. 24) A. True B. False	
15.	Which of the following is NOT one of the suggested "R" principles of Alzheimer's care? (p. 26)  A. Rituals should be maintained  B. Redirect away from repetitive thought patterns  C. Restrict unusual behaviors  D. Respect the person	
16.	is a home based therapeutic intervention that matches activities to cognitive and functional capabilities. (p. 30)  A. Interceptive cognitive conditioning  B. Tailored activity program  C. Restorative behavioral modeling  D. Reflective functional mirroring	
17.	The water heater should be set to 150 degrees to avoid scalding. (p. 35) A. True B. False	
18.	A person with AD may mistakenly believe that shadows on the wall are really people in the room with them. This is an example of: (p. 39)  A. Hallucination  B. Adaptation  C. Illusion  D. Delusion	
19.	Which of the following is TRUE regarding how AD affects the senses? (p. 40-42) A. decreased ability to detect color B. loss or decrease in ability to smell C. hypersensitivity to taste D. diminished hearing	
20.	Stigma is the use of negative labels to identify a person living with a disability or illness. (p. 45) A. True B. False	
	c72011g3417r41120t72011	