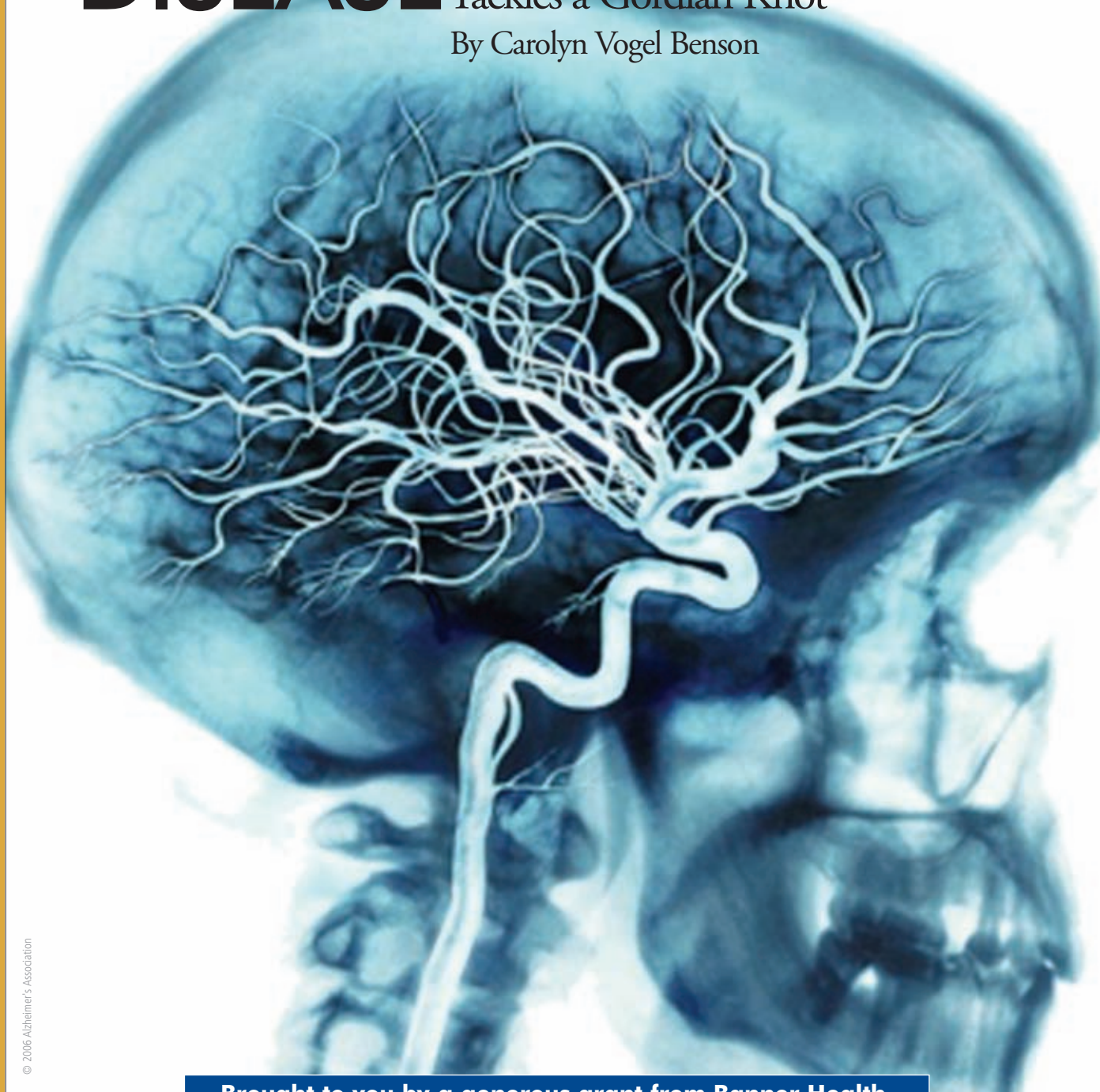


UNTANGLING ALZHEIMER'S DISEASE

Modern Science
Tackles a Gordian Knot

By Carolyn Vogel Benson



A mind *is* a terrible thing to waste.

For at least 15 years, my mother's brain, attacked by Alzheimer's disease, wasted away until she had very little memory left and could not walk or eat. Beginning in 1989, two years after her triple bypass operation, I noticed that my 67-year-old-mother found it difficult to use an instamatic camera while we were vacationing in France. At home, she had slight memory problems and dropped out of several volunteer activities.

At first, my sisters and I thought her memory problems were due to aging, but a few years later, while we were visiting, we realized something was very wrong: She had been eating cold food from the refrigerator because she couldn't operate the microwave or the stove.

Shopping for food or clothes was no longer possible unless we accompanied her. Even then, she had difficulty deciding what to buy. She became more and more dependent upon my sister, Charlotte, who lived in the same town.

My mother increasingly became more and more isolated. In retrospect, we think she was hiding the limitations that Alzheimer's disease imposed on her verbal skills and her

memory. She wanted only close family members around her. She refused opportunities to socialize, even with trusted old friends—women she had known for 50 to 60 years, women my sisters and I called "aunt."

In the summer, neighbors noticed that my mother's door was open at 1 a.m., and she was asleep in a chair with the television on. In her isolation, my mother became a chain smoker, and we feared she would perish in a fire. When we tried to arrange for housekeepers or professional caretakers to help her in her home, she adamantly refused for fear they would steal from her or worse.

Because my youngest sister and I live in other states and Charlotte, who lived near my mother, was developing a progressive neuromuscular disease, we moved our mother to an assisted living facility with many amenities and services.

For awhile, she seemed to be improving, but then she began to retreat to her room, telling us she didn't like any of the residents. Doctors finally made the diagnosis of Alzheimer's disease when an MRI of her brain showed a thinning of the cerebral cortex, the layer of grey matter normally filled with thousands of nerve cells.

Her memory and behavior became much worse, exacerbated by a mini-stroke that further injured her brain. She told people she was 50 years old when she was actually 80. She complained to the residents that we had stolen her good jewelry. She was critical of many people, lost her temper easily, and once tried to hit Charlotte with her cane. Aides had to fetch her to take her to the dining room; she couldn't remember where it was.

As the mini-strokes and other health problems continued, we found a good nursing home that specialized in the care of Alzheimer's patients. By then, she was unable to walk and was incontinent. She became less verbal, at times staring off into the distance or vacantly looking at her outspread hands. Eventually, she refused to eat and drink. She was operated on for gallstones and her dementia increased dramatically. Two weeks later, we agreed that she needed hospice care.

My mother died from Alzheimer's disease and vascular dementia in April 2006 at the age of 83. That year, she was one of 4.5 million Americans with the disease.

'AN INDISCRIMINATE KILLER OF MIND AND LIFE'

Alzheimer's disease (AD) is the most common form of dementia, a group of conditions that progressively destroy brain cells, causing a decline in mental function. In AD, a normal biochemical process goes awry, causing the brain to become filled with tangles of neurons and sticky plaques consisting of a substance called beta-amyloid. As the disease progresses, connections between the brain's nerve cells are impaired or lost and the cells die.

In vascular dementia, the second-most-common form, arteriosclerosis reduces blood flow to the brain's nerve cells. Cells in the affected regions die because of lack of oxygen or hemorrhaging from weakened blood-vessel walls. Alzheimer's disease and vascular dementia can occur together in some people, as they did in my mother.





ALZHEIMER'S IS NOT NORMAL AGING

The abnormal brain pathology of AD has been known since 1906, when Dr. Alois Alzheimer, a German psychologist and histopathologist, performed an autopsy on a 55-year-old patient who had suffered for years from a progressive loss of brain function. Alzheimer observed that the patient's cerebral cortex was thinner than normal and contained numerous tangles and "senile" plaques. The cerebral cortex controls conscious thought, mental activity, and voluntary movements. It also processes sensory information that reaches the brain from the environment.

Senility—the loss of memory and gradual impairment of thinking, reasoning, and verbal skills—was once thought to be a normal part of aging, but Alzheimer realized that this was not normal for a 55-year-old. He had discovered an early-onset form of the disease that later was named after him.

As medical science improved and learned more about disease processes that occur with

aging, it became apparent that not all seniors become senile or suffer heart attacks and strokes. In the normal aging brain, some plaques and tangles develop, but not to the extent seen in AD. Also, people usually do not lose neurons in large numbers. In fact, neurons are programmed to last 100 years or more.

People differ in how their brains react to the changes that occur in normal aging. Cognitive functions may slow down, but, given enough time, most people who are aging normally can perform complex tasks of attention, learning, and memory as well as younger people.

Just as heart attacks and strokes are the end result of arteriosclerosis that begins early in life, cascades of abnormal biochemical events in the brain begin many years before the signs and symptoms of AD or other dementias become noticeable. Although these diseases have a strong genetic component, lifestyle and environment are thought to be contributing factors.

Initially, people with AD experience memory loss and confusion. As the disease progresses, thinking and reasoning become difficult. In time, decision-making and language skills become impaired, and personality and behavior changes occur, with anxiety, suspiciousness, or agitation becoming common or frequent. Delusions and hallucinations may also develop. A severe loss of mental function is the end result.

Most people who die from Alzheimer's disease have had years of cognitive decline—years in which they could have traveled, learned new things, developed deeper relationships with family and friends.

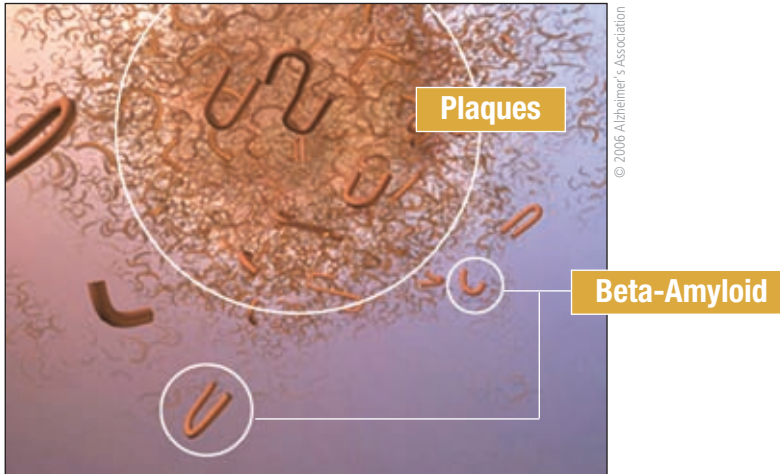
In 1994, six years after leaving the White House and retiring to his ranch, President Reagan wrote a letter to the nation disclosing that he had AD, the disease he once described as "an indiscriminate killer of mind and life." In one speech, he noted that Alzheimer's disease "ranks among the most severe of afflictions, because it strips people of their memory and judgment and robs them of the essence of their personalities. As the brain progressively deteriorates, tasks familiar for a lifetime, such as tying a shoelace or making a bed, become bewildering. Spouses and children become strangers."

The disease Reagan described while in office greatly affected the course of his life in retirement. He died of Alzheimer's disease in 2004. In his honor, both houses of the 108th Congress sponsored a bill, the Ronald Reagan Alzheimer's Breakthrough Act of 2004 (S. 2533 and HR 4595). This bill died in committee.



THE BRAIN INTERRUPTED

Brains of persons with AD have three distinguishing characteristics: insoluble beta-amyloid plaques, neurofibrillary tangles, and gradual atrophy (shrinkage).



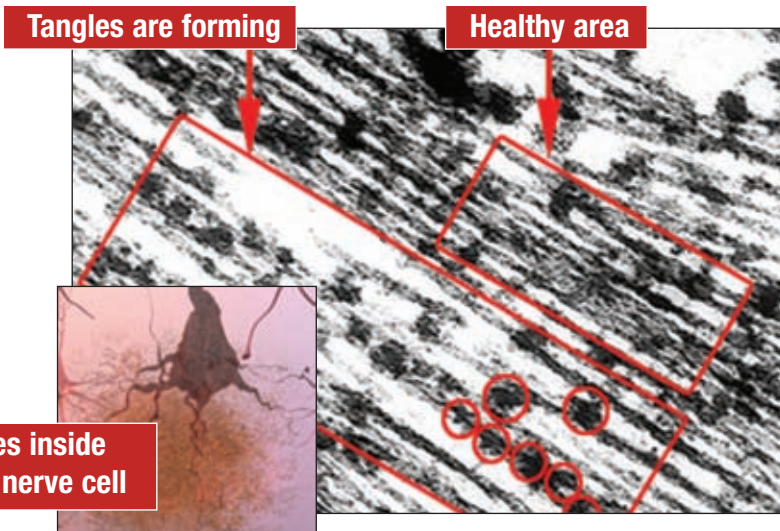
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BETA-AMYLOID PLAQUES

The process by which the beta-amyloid protein abnormally accumulates and forms plaques begins with a protein called amyloid precursor protein or APP, which is produced inside the neuron and normally embeds in the neuron's cell membrane. APP is cleaved into fragments or peptides by three enzymes (proteins that cause a chemical reaction). The APP peptide is necessary for the neuron's growth and maintenance. If the enzymes snip APP in the wrong order or cleave it in the wrong places, beta-amyloid is released into the spaces between neurons, where it sticks to other beta-amyloid peptides from other neurons that have undergone the same process. In response, the immune system releases inflammatory cells that normally degrade and remove damaged neurons, foreign substances, and plaques. Instead, these responders appear to be engulfed by the sticky plaque.

NEUROFIBRILLARY TANGLES

Scientists believe that the brain's neurons disintegrate when they no longer have sufficient quantities of the beta-amyloid peptide necessary for cell maintenance. In the cascade of events that follows, the protein that provides support for the neuron's microtubules, called tau, forms abnormally into long threads that wind around each other in a helix shape and accumulate within neurons. Without the support of tau, the microtubules disintegrate and can no longer transport nutrients and other materials down the neuron to the end of its axis and back. Messages that neurons transport are also interrupted. The neuron is unable to transmit electrical charges to the synapses, where special chemicals called neurotransmitters carry the message to another neuron. Gradually the connections between neurons are lost and they die.



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BRAIN ATROPHY

The abnormal pathology of Alzheimer's disease begins in the brain's hippocampus, the center for memory and spatial navigation. Humans actually have two hippocampi, located on the underside of the brain, beneath each temporal lobe. In early AD, the hippocampi shrink. As AD progresses, other areas of the brain are affected. The death of numerous neurons causes the brain to shrink in size.

ABNORMAL GENES

Scientists have discovered three gene variants that cause early-onset AD, a disease that runs in families. So far, they have also discovered two susceptibility genes for late-onset AD. Abnormal variants of genes for both types of AD cause the production of toxic levels of certain beta-amyloid peptides.

Cardiology researchers first discovered one of the normal genes, ApoE, and its protein, apolipoprotein, in 1993. Apolipoprotein removes cholesterol from the blood. However, if it is produced by a mutation of the gene, designated ApoE-4, the apolipoprotein becomes an active participant in coronary artery disease.

ApoE-4 has also been found to be the main culprit in both early-onset (familial) and late-onset Alzheimer's disease. ApoE-4 is found in 40 percent of people with the disease, and it appears to accelerate the disease process. People who have had a brain injury are more prone to developing AD if they have this gene.

Discovery of SORL1, the second susceptibility gene for Alzheimer's, was announced in January 2007. The study that led to the discovery was important because it included people of diverse ethnic and racial backgrounds, whereas the ApoE studies were of Caucasians only.

Researchers determined that people with AD have less than half the amount of the normal SORL1 protein in their blood cells than do healthy people from the same groups. In the laboratory, low levels of this protein were found to increase production of the beta-amyloid fragments found in plaque, while high levels decreased production. Discovering which of the 500 variants of SORL1 causes AD is the next challenge.

Scientists suspect that there are at least three to seven susceptibility genes involved in Alzheimer's. None of the known genes by themselves appears to be the sole cause.

DIAGNOSING AND TREATING AD

AD can go unnoticed for a very long time. Quite often, people with the disease attempt to hide it. If no one expresses concern to a doctor,

the disease may continue to go unnoticed until the person can no longer function or depression or paranoia set in.

Although many clinicians can diagnose it with some degree of accuracy by performing a physical exam and conducting mental-function tests, AD can be confused with other dementias. Available technologies such as MRIs usually are unable to help with a diagnosis until brain atrophy is apparent, which happens late in the disease. A definitive diagnosis of AD can be made only through a brain autopsy.

However, new neuroimaging techniques are being developed that will soon be able to help clinicians make an early diagnosis. For example, studies using PET (positron emissions tomography) have discovered low rates of glucose metabolism in certain regions of the cerebral cortex of people who have AD.

In a recent study, investigators performed PET and magnetic resonance imaging (MRI) scans on healthy participants who were given neuropsychological tests. The participants did not differ significantly in age, gender, education, or neuropsychological test scores; however, half carried the mutant ApoE-4 gene. Results of this watershed study confirmed previous findings: AD changes occur in cognitively healthy young adults. If the changes in glucose metabolism are validated by further studies that track brain changes over time, treatment before symptoms occur will be possible.

Two new imaging compounds have proven useful in highlighting tangles and plaques in the brains of persons with AD, and they may prove to be useful in imaging studies to detect early signs of the disease. "We urgently need techniques to see brain changes in the earliest stages of cognitive decline, so that we can identify people at risk and test drugs to stop or slow progression of Alzheimer's," says Richard H.odes, M.D., director of the National Institute on Aging (NIA) of the National Institutes of Health. NIA launched its Alzheimer's Disease Neuroimaging Initiative last year.

Biomarkers of AD in blood, urine, and cerebrospinal fluids are also critical for making an accurate early diagnosis and may be a valuable tool for gauging the impact of certain treatments. So far, scientists have identified 23 biomarkers in cerebrospinal fluid. A study is currently being conducted testing levels of an abnormal tau protein, the protein involved in neurofibrillary tangles, in cerebrospinal fluid, to determine whether it could be an early biomarker for AD.

Several drugs are on the market to treat early

and middle stages of Alzheimer's disease. These drugs inhibit the excessive breakdown of the neurotransmitter acetylcholine. By doing so, they moderately reduce the severity of symptoms. Another drug, with an entirely different mode of action, has been approved for patients with severe symptoms. None of these drugs slows down disease progression or provides a cure.

Support groups for people with early-stage AD are important to help them understand the disease and deal with it, find helpful resources, and plan for the future. The Alzheimer's Association, NIA-funded Alzheimer's Disease Centers (ADCs), and other organizations have established several types of early-stage support groups at centers throughout the United States. ADCs are also developing comprehensive, individualized management approaches to help home-dwelling patients.

CAN ALZHEIMER'S BE PREVENTED?

Risk factors are traits and lifestyle habits that increase the chances of getting a disease. A risk factor for a disease does not necessarily mean a person will develop it, and some risk factors can be reduced or prevented.

AGE

According to the National Institute on Aging, the risk for developing AD doubles every five years after 65. Several studies estimate that more than half the people older than 85 have the disease. This is significant because more than 34 million Americans are now 65 or older, and the group at highest risk—those 85 and older—are the fastest-growing population group in the United States.

FAMILY HISTORY, GENETICS

A study of identical twins age 65 and over in the Swedish Twin Registry found that genetic inheritance explained about 80 percent of Alzheimer's risk. Environmental factors that the twins did not share explained the other 20 percent. However, genes are not destiny. If AD appeared in one twin, the other did not necessarily develop it; among those who did develop it, for some, it happened as much as 16 years later. The study found that, for male identical twins, the risk of developing AD if the other twin had it was 45 percent. For females, the risk of developing AD if the other twin had it was 60 percent, but that is because women live longer than men.

According to the NIA, a child who has a parent with one of the three mutant genes known to cause early-onset AD has a 50-50 chance of

inheriting that gene and developing early-onset AD.

HIGH CHOLESTEROL AND HOMOCYSTEINE

Basic science research in laboratories, population studies, and animal research suggests a link between high cholesterol and Alzheimer's disease. Some research indicates that statins, drugs that lower cholesterol, may slow the progression of AD. Other studies do not support this theory. Further research on larger populations is planned.

Homocysteine, another substance associated with heart disease, may also be a risk factor for AD. Laboratory studies have demonstrated that high levels of this amino acid can cause neurons to stop functioning and die. Folic acid and vitamins B6 and B12 reduce high blood levels of homocysteine.

Eating cold-water fish two or three times a week could be good for the brain as well as the heart. Good sources of B6 and B12 are oily fish and clams. Excellent sources of folic acid are green, leafy vegetables; asparagus; Brussels sprouts; broccoli; oranges and grapefruits; and fortified cereals. Older people are generally deficient in B6 and B12 and may need supplements. (Discuss supplements of any vitamins and herbals with a physician before taking them; certain dosages can be toxic and harmful drug interactions could occur.)

HIGH BLOOD PRESSURE

High blood pressure that begins in mid-life has been associated with AD. When the heart pumps blood out of its chambers, the force applied against the walls of the arteries is called "blood pressure." The intensity of the force, the amount of blood pumped, and the size and flexibility of the arteries all determine blood pressure. High blood pressure, which often goes unnoticed, can injure the walls of the arteries.

Some studies have reported that the risk for AD is reduced when blood pressure is reduced. More studies are needed to further explore this possible risk factor. Several blood pressure drugs are being investigated for their ability to prevent or slow the progression of AD. Inderol and potassium-sparing diuretics appear to have beneficial effects.

GLUCOSE METABOLISM: INSULIN RESISTANCE AND TYPE-2 DIABETES

Insulin resistance is a condition in which the body produces insulin, but cells do not use it correctly. The pancreas produces insulin, which helps cells take in glucose and convert it to

energy. Too much insulin in the blood causes inflammation and oxidative stress on cells—two factors that contribute to neuronal damage. Being obese or overweight affects the way insulin works in the body. Extra fat tissue can make the body resistant to the action of insulin, but exercise helps insulin work well.

Insulin resistance leads to type-2 diabetes, a condition that develops in adults when levels of blood glucose become elevated because the body produces insufficient insulin. Type-2 diabetes and AD have several characteristics in common: increasing prevalence with age; genetic predisposition; and deposits of two different types of a toxic amyloid protein, one in the brain of someone with AD, the other in the pancreas of someone with type-2 diabetes.

A diet low on the glycemic index and regular exercise can prevent or reduce insulin resistance and type-2 diabetes.

The NIA is funding several clinical trials to investigate whether managing glucose, blood pressure, and blood lipids such as cholesterol will have an effect on the brain structure and cognitive function of people with AD. Two drugs will be tested—one that makes cells more sensitive to insulin and an insulin nasal spray.

INFLAMMATION

Inflammatory cells are present in the brains of people with AD, but whether they are present as an immune response or as causative factors is unknown. One study did find a correlation between brain infection with the herpes simplex (cold sore) virus and AD, which may account for chronic inflammation. Population studies of nonsteroidal anti-inflammatory drugs suggest they may reduce AD symptoms, but clinical trials so far have not supported this.

Ibuprofen is being studied for its effects on the progression of AD in a clinical trial that will look at biomarkers of the disease in blood, cerebrospinal fluid, and urine.

Curcumin, a yellow substance found in the spice turmeric, has been discovered to have anti-inflammatory, anti-cholesterol, and anti-oxidant properties. A clinical study will test whether this substance can prevent or treat AD.

OXIDATIVE STRESS/DIET

Oxidative stress on nerve cells results from molecules called free radicals, which are normal byproducts of cell metabolism. When free radicals accumulate excessively in cells, they die. Free radical damage accumulates with age.

The body's immune system can also produce free radicals to neutralize viruses and bacteria. Pollution, radiation, cigarette smoke, and

herbicides stimulate their production as well. Under normal circumstances, the body can eliminate free radicals if anti-oxidant are available.

Some laboratory studies indicate that anti-oxidants from food and supplements may provide protection against oxidative damage. Eating five to eight servings per day of anti-oxidant-rich fruits and vegetables can benefit the brain (and the heart as well). Top on the list of anti-oxidant-rich foods are small red beans, wild blueberries, red kidney beans, pinto beans, cultivated blueberries, artichokes, raspberries, blackberries, pecans, and apples.

Studies of several possible anti-oxidant treatments have been concluded and new studies are underway. DHA, an omega-3 fatty acid abundant in some fish, is being tested as a possible treatment for AD because it has anti-oxidant and neuroprotective mechanisms. Vitamins E and C, as well as alpha-lipoic acid and coenzyme Q, are also being evaluated.

Sage, an herb rich in anti-oxidants, improves memory recall in young adults, according to recent research. Sage is believed to reduce anticholinesterase, an enzyme that breaks down the important neurotransmitter acetylcholine; low levels of acetylcholine have been observed in people with AD. A clinical study will test the effects of sage on memory and mental performance in AD patients.

Huperzine A, a derivative of a Chinese herb rich in anti-oxidants and a natural cholinesterase inhibitor, is also being studied as a possible intervention in AD.

HORMONES

Reduction in hormones may contribute to AD in older people. Estrogen has a positive effect on the brain, but, according to one study, it does not slow progression in those who already have the disease. Also, women older than 65 who are given estrogen are actually at increased risk of developing dementia. However, scientists believe that some forms of estrogen given earlier than age 65 may be beneficial. The therapeutic potential of estrogen for improving memory and for helping postmenopausal women with AD to live independently is being examined in a clinical trial sponsored by the NIA.

Treatment with testosterone improves performance on tests of spatial ability in men with low testosterone levels and AD. This means that some men with AD who receive testosterone may be able to navigate their environment better without getting lost or injured. Trials of testosterone are also being conducted.

BRAIN TRAUMA

Large numbers of people who have bypass surgery have complained that their minds are less sharp and memories not as keen as they were before surgery. Some recover, but others do not. One study gave patients memory tests before and after heart surgery; results showed that patients with the AD-causing ApoE-4 variant were more likely to suffer greater memory impairment.

Boxers, football players, and other athletes who have received frequent or multiple blows to the head also have a greater risk of moderate to severe brain damage at a younger age if they carry the ApoE-4 gene.

ANESTHETICS

One of the most commonly used anesthetics, isoflurane, has been associated with development of beta-amyloid plaques in the brain after surgery. When scientists applied the anesthetic to nerve cells in a culture, the beta-amyloid protein was produced and led to cell death. Scientists warn that isoflurane should be used with caution in older people.



LACK OF MENTAL, SOCIAL, AND PHYSICAL ACTIVITY

Mental Activities—According to one study, playing chess once a week may reduce the risk of dementia by about 7 percent; playing three times a week may reduce it by 21 percent. Writing, solving crossword puzzles and sudoku, or playing board games all seem to help. Several studies suggest that people who pursue intellectually challenging work throughout their lives have a much lower risk of AD.

A study of transgenic mice (mice that had been given the human AD gene) published in the January 24, 2007, edition of the *Journal of Neuroscience*, reported that learning slowed the build-up of plaques and tangles in the mice's brains.

Social Activities—Loneliness is linked to a

UNTANGLING ALZHEIMER'S DISEASE

higher risk for AD. A number of studies of animals and older people link a high level of social engagement to a significant reduction in cognitive decline.



Exercise—Regular exercise is important to staying healthy at any age. A study using MRIs to measure brain activity in healthy adults aged 58 to 78 before and after a six-month program of brisk walking showed increased functioning in certain areas of the brain, as well as improved cardiovascular fitness. Compared to the group that was inactive, walkers were more attentive and were able to focus on goals without being distracted by unimportant information.

It is not known whether physical, mental, and social activity can prevent cognitive decline or delay the development of AD, but there are signs these activities could make a difference.

Although research indicates that people with AD age 60 and older experience a decline in new learning, many with mild symptoms of the disease are able to carry on with their lives, especially when given assistance, support, and understanding.

Molly Picon, beloved star of Yiddish theater, Broadway, and films, developed AD when she was in her late 70s. In 1979 at the age of 83, she took her autobiographical show, "Hello, Molly," on the road in an effort to stay connected

to her audiences. In 1982, she acted in a short film about dementia, "Grandma Forgot to Wave Back." Picon died of Alzheimer's disease 10 years later at the age of 93.

ADDITIONAL RESEARCH

The NIA has declared that Alzheimer's disease is an urgent national research priority and has funded 32 ADCs throughout the country that are focusing on translating research advances into improved diagnosis and care, and finding interventions that will prevent and cure AD. One major advantage of ADCs is that they provide a network for sharing new ideas and research results.

In addition to the studies mentioned above, several drugs and vaccines are also being tested. A vaccine against beta-amyloid plaque was developed several years ago and was successful in transgenic mice. A human safety trial of the vaccine was successful as well, but the Food and Drug Administration halted the next trial when 5 percent of patients developed encephalitis—an inflammation of the brain triggered by the body's immune response. The drug company is refining the vaccine for future trials.

THE FUTURE

This year, according to the Alzheimer's Association, 4.9 million Americans over 65 are living with AD, and between 200,000 and 500,000 under 65 are living with early-onset AD and other dementias. Researchers at the NIA estimate that, by 2050, 13.2 million Americans will have AD.

These prevalence trends imply an enormous impact on large numbers of people: those with the disease, caregivers, and family and friends. Needless to say, the future of our nation's health-care system will be in jeopardy if methods to prevent, slow down, or cure Alzheimer's disease are not found within the next few years.

Although medical science may be "on the right track," the train needs to go much faster. More funding to fuel it will help.

THE ALZHEIMER'S ASSOCIATION

24-hour helpline: 1-800-272-3900

E-mail: info@alz.org

Website: www.alz.org

The nonprofit association has up-to-date information on research, treatments, publications, suggestions on how to live with Alzheimer's, locations of chapters, and resources on how to find care. For a good understanding of the pathology of AD, take its Inside the Brain Interactive Tour.

ALZHEIMER'S DISEASE EDUCATION AND REFERRAL (ADEAR) CENTER

1-800-438-4380

E-mail: adear@nia.gov

Website: www.alzheimers.nia.nih.gov

The NIA's ADEAR Center provides a variety of information on AD, including information about care-giving, diagnosis and treatment, and results of research findings. Several booklets are available to the public. The ADEAR Center also has a database of clinical trials, develops recommended reading lists, and provides referrals to local AD resources.

ELDERCARE LOCATOR

1-800-677-1116

Website: www.eldercare.gov

The Administration on Aging provides information about community resources available from its agency.

PREVENTIVE GENES

Some genes may actually help prevent Alzheimer's disease or vascular dementia.

ApoE-2, a rare variant of the apolipoprotein gene, may provide protection against Alzheimer's disease.

The gene CETP VV, which has been found in some centenarians, prevents arteriosclerosis and slows aging. The discovery was made in December 2006 after researchers studied 158 people of Ashkenazi Jewish descent, ages 75 to 85, for eight years and recorded which participants were diagnosed with dementia. Those who never developed dementia were five times as likely to have the CETP VV gene as those who had dementia. Although researchers don't know how the gene protects seniors, they do know from previous studies that it could affect the size of blood lipoproteins that either deposit cholesterol or clear it away.

ALZHEIMER'S ASSOCIATION: COMMITTED TO A WORLD WITHOUT ALZHEIMER'S

The Alzheimer's Association is the leading voluntary health organization in Alzheimer's research, care, and support. Our mission is to eliminate Alzheimer's disease through the advancement of research; to provide and enhance care and support for all affected; and to reduce the risk of dementia through the promotion of brain health.

Recently the Alzheimer's Association released a report that shows there are currently more than 5 million people in the United States living with Alzheimer's disease. It is now the seventh leading cause of death in the country and the fifth leading cause for those over age 65. Alzheimer's disease has a tremendous impact on people with the disease, their families and those who care for them, the government, and society as a whole. If the disease continues on its current path, experts estimate that there will be as many as 16 million Americans living with the disease by 2050.

However, there is hope. There are currently nine drugs in Phase III clinical trials for Alzheimer's, several of which show great promise to slow or stop the progression of the disease. This, combined with advancements in diagnostic tools, has the potential to change the landscape of Alzheimer's.

In addition to providing information, care, and services for those touched by the disease, the Alzheimer's Association has been a catalyst behind nearly every major breakthrough and advance in Alzheimer's research since 1982. With your help, we can help change the course of Alzheimer's. To learn what you can do to join us in our fight against this devastating disease, visit www.alz.org.

*Harry Johns
President and CEO
Alzheimer's Association*

B'NAI B'RITH AND ALZHEIMER'S DISEASE

B'nai B'rith International became involved with Alzheimer's (AD) through its sponsorship of senior housing. This is a disease that affects entire families. While a spouse or adult child cares for a loved one with Alzheimer's, that loved one will forget the family's faces. Even Alzheimer's patients in relatively good physical shape may require care to protect them from dangers they are no longer aware of. This places a burden on families that is both emotional and financial.

The disease is all-encompassing. Developments in diagnosis, treatment, and prevention must be pursued. We are closer to effective management, but we are not there yet.

B'nai B'rith will continue to advocate for funding for research. We must also help families plan and coordinate care. This means making sure that governments, doctors, and families themselves recognize the importance of family caregivers and the challenges they face. It is crucial that we reach out to these families.

There are options and services available for adapting homes and to provide respite to caregivers. One focus of B'nai B'rith's Solving the Aging-in-Place Puzzle Initiative is on getting families to talk together about what the future may hold. This can make it easier to deal with AD (or any other disease) as it begins to change the way a loved one moves, thinks, remembers, and lives.

B'nai B'rith is committed to battling AZ because this is one of the most devastating and prevalent illnesses in the senior population. This is a disease that overwhelms and isolates. Improved treatment and a cure are possible, but only if we push. This involves spreading the word about awareness, support, and community. B'nai B'rith International pledges to continue the fight.

*Rosalind Klein
Chair, B'nai B'rith International
Senior Advocacy Initiative*

BANNER'S COMMITMENT TO THE FIGHT AGAINST ALZHEIMER'S

Alzheimer's disease afflicts 10 percent of everyone over age 65 and almost half of everyone over 85. As more people live to older ages, the number of afflicted patients is expected to soar. Treatments to address and end Alzheimer's are urgently needed to avert an overwhelming public health problem. The Banner Alzheimer's Institute (BAI) was recently established to address this problem in the shortest time and most meaningful way possible. Our goals are to end Alzheimer's without losing a generation, while setting a new standard of care for patients and families along the way. Here's how we hope to accomplish these goals.

World-leading clinical trials and brain imaging programs will evaluate promising disease-slowing medication and vaccine therapies in the most rigorous, rapid, and cost-effective way. Furthermore, brain imaging techniques will be used to evaluate promising prevention therapies in cognitively normal carriers of a common Alzheimer's susceptibility gene, without having to study thousands of people or wait many years to see if they develop symptoms. Our goal is to identify effective disease-stopping and prevention therapies within the next 12 years.

Meantime, specialists in our Memory Disorders Clinic provide highly personalized state-of-the-art evaluation and treatment services, and specialists in our Family and Community Services Program help address the non-medical needs of our patients and their families to the fullest extent possible. Working together, they intend to prove to third-party payers—and everyone else—why this comprehensive service should become the new standard of care.

It is time to end this dreadful disease. With B'nai B'rith's support, we and our colleagues can do just that.

*Eric M. Reiman, M.D.
Executive Director
Banner Alzheimer's Institute*