A REVIEW ARTICLE ON ALZHEIMER’S DISEASE

¹Feroja Easmin*, ¹Md. Habibur Rahman, ¹Md. Mizanur Rahman, ¹Ripa Moutoshi, ¹Rajib Ghose, ²Samira Yesmin, ³Asma Alam Chowdhury

¹Southeast University, BANGLADESH
²University of Development Alternative, BANGLADESH
³East-West University, BANGLADESH

Abstract
Alzheimer’s disease is a devastating disease in humans associated with significant morbidity and mortality. As a country of the Asia Pacific region Bangladesh is considered to be a risk country for developing Alzheimer’s disease. This study represents the overview of Alzheimer’s disease in Bangladesh and in present world. Alzheimer’s disease is an irreversible, progressive brain disorder that slowly destroys memory and thinking skills and, eventually, the ability to carry out the simplest tasks. In most people with Alzheimer’s, symptoms first appear in their mid-60s. Alzheimer’s disease is the most common cause of dementia among older adults. Alzheimer's disease is a progressive and neurodegenerative disorder which involves multiple molecular mechanisms. Alzheimer's disease is the sixth-leading cause of death in the United States and the only cause of death among the top 10 in the United States that cannot be prevented, cured or even slowed. 35 million people worldwide have Alzheimer’s disease. About 4.5 million Americans suffer from this condition, which usually begins after age 60. According to Alzheimer Society of Bangladesh, The number of dementia patients in 2015 is about 4 lakh 60 thousand, which will be turned into 8 lakh 34 thousand in 2030, and in 21 lakh 93 thousand if not prevented. Currently there is no cure for this devastating disease. But drug and non-drug treatments may help with both cognitive and behavioral symptoms. Researchers are looking for new treatments to alter the course of the disease and improve the quality of life for people with dementia.

Keywords: Alzheimer’s disease, Brain disorder, Dementia, Neurodegenerative disorder, Cognitive and behavioral symptoms.

Corresponding Author:
Feroja Easmin
Southeast University
BANGLADESH

E-mail: pharmacisthabib@gmail.com
Phone: +8801965266177

Available online: www.ijipsr.com June Issue
INTRODUCTION

Alzheimer's disease is the sixth-leading cause of death in the United States and the only cause of death among the top 10 in the United States that cannot be prevented, cured or even slowed. 35 million people worldwide have Alzheimer’s disease. The most common form of dementia among older people is Alzheimer's disease. About 4.5 million Americans suffer from this condition, which usually begins after age 60. One in eight older Americans has Alzheimer's disease [1].

Dementia is not a disease itself. It's a group of symptoms that are caused by various diseases or conditions. Read how dementia develops, what causes it, and which conditions are treatable.

More than 15 million Americans provide unpaid care valued at $210 billion for persons with Alzheimer's and other dementias. Payments for care are estimated to be $200 billion in the United States in 2012.

The disease is named after Dr. Alois Alzheimer. In 1906, Dr. Alzheimer noticed changes in the brain tissue of a woman who had died of an unusual mental illness. Her symptoms included memory loss, language problems, and unpredictable behavior. After she died, he examined her brain and found many abnormal clumps (now called amyloid plaques) and tangled bundles of fibers (now called neurofibrillary, or tau, tangles).

These plaques and tangles in the brain are still considered some of the main features of Alzheimer’s disease. Another feature is the loss of connections between nerve cells in the brain. Neurons transmit messages between different parts of the brain, and from the brain to muscles and organs in the body. Although treatment can help manage symptoms in some people, currently there is no cure for this devastating disease.
History of Alzheimer’s disease

In 1901 a German psychiatrist Alois Alzheimer identified the first case of what became known as Alzheimer's. A fifty year old woman he called her Auguste D had Alzheimer's. She died in 1906 of the disease. It was first described as a distinctive disease by Emil Kraepelin [2].

He included Alzheimer's disease, also named presenile dementia by Kraepelin, as a subtype of senile dementia in the eighth edition of his Textbook of Psychiatry, published in 1910. For most of the 20th century, the diagnosis of Alzheimer's disease was reserved for individuals between the ages of 45 and 65 who developed symptoms of dementia. It led to the diagnosis of Alzheimer's disease independently of age.

The terminology changed after 1977 when a conference on AD concluded that the clinical and pathological manifestations of presenile and senile dementia were almost identical, although the authors also added that this did not rule out the possibility that they had different causes [3].

The term senile dementia of the Alzheimer type (SDAT) was used for a time to describe the condition in those over 65, with classical Alzheimer's disease being used for those younger. Eventually, the term Alzheimer's disease was formally adopted in medical nomenclature to describe individuals of all ages with a characteristic common symptom pattern, disease course, and neuropathology [4].

Epidemiology

Two main measures are used in epidemiological studies: incidence and prevalence. Incidence is the number of new cases per unit of person–time at risk (usually number of new cases per thousand person–years); while prevalence is the total number of cases of the disease in the population at any given time [5].
Regarding incidence, cohort longitudinal studies (studies where a disease-free population is followed over the years) provide rates between 10 and 15 per thousand person–years for all dementias and 5–8 for AD, which means that half of new dementia cases each year are AD. Advancing age is a primary risk factor for the disease and incidence rates are not equal for all ages: every five years after the age of 65, the risk of acquiring the disease approximately doubles, increasing from 3 to as much as 69 per thousand person years. There are also sex differences in the incidence rates, women having a higher risk of developing AD particularly in the population older than 85. The risk of dying from Alzheimer's disease is twenty-six percent higher among the non-Hispanic white population than among the non-Hispanic black population, whereas the Hispanic population has a thirty percent lower risk than the non-Hispanic white population. [6]

Alzheimer's disease causes loss of brain cells in areas of the brain. Some of the deterioration may be related to a loss of chemical messengers in the brain, called neurotransmitters that allow nerve cells in the brain to communicate properly. People with Alzheimer's disease have two things in the brain that are not normal: amyloid plaques and neurofibrillary tangles.

The cause for most Alzheimer's cases is still mostly unknown except for 1% to 5% of cases where genetic differences have been identified. Several competing hypotheses exist trying to explain the cause of the disease:

**Sign and Symptoms of Alzheimer's disease** [7]

Memory loss is usually the first sign of Alzheimer's disease. Having some short-term memory loss in your 60s and 70s is common, but this doesn't mean it's Alzheimer's disease.

Compare these examples of normal memory problems and the types of memory problems that may be caused by Alzheimer's disease.

### Table 2: Symptoms of normal forgetfulness versus Alzheimer's disease

<table>
<thead>
<tr>
<th>In normal forgetfulness, the person may forget:</th>
<th>In Alzheimer's disease, the person may forget:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Parts of an experience.</td>
<td>An entire experience</td>
</tr>
<tr>
<td>Where the car is parked.</td>
<td>What the car looks like.</td>
</tr>
<tr>
<td>A person's name, but remember it later.</td>
<td>Ever having known a particular person.</td>
</tr>
</tbody>
</table>
Alzheimer’s disease also causes changes in thinking, behavior, and personality. Close family members and friends may first notice these symptoms, although the person may also realize that something is wrong.

Following are some of the symptoms of the different stages of Alzheimer's disease. They vary as the disease progresses. Talk to your doctor if a friend or family member has any of the signs.

**Disease presentation**

Every person with Alzheimer’s disease experiences the disease differently, but patients tend to experience a similar trajectory from the beginning of the illness to its merciful end. The precise number of stages is somewhat arbitrary.

AD progresses gradually and can last for decades. There are four stages of the disease, each with its own challenges and symptoms. By identifying the current stage of the disease, physicians can predict what symptoms can be expected in the future and possible courses of treatment. Each case of AD presents with a unique set of symptoms, varying in severity.  [8]

![Fig.3: Brain Atrophy in Alzheimer’s disease](image)

**Pre-dementia**

The first symptoms are often mistakenly attributed to ageing or stress. Detailed neuropsychological testing can reveal mild cognitive difficulties up to eight years before a person fulfills the clinical criteria for diagnosis of AD. These early symptoms can affect the most complex daily living activities. The most noticeable deficit is short term memory loss, which shows up as difficulty in remembering recently learned facts and inability to acquire new information [9]. Subtle problems with the executive functions of attentiveness, planning, flexibility, and abstract thinking, or impairments in semantic memory (memory of meanings, and
concept relationships) can also be symptomatic of the early stages of AD. Apathy can be observed at this stage, and remains the most persistent neuropsychiatric symptom throughout the course of the disease. Depressive symptoms, irritability and reduced awareness of subtle memory difficulties are also common [10]. The preclinical stage of the disease has also been termed mild cognitive impairment (MCI). This is often found to be a transitional stage between normal ageing and dementia. MCI can present with a variety of symptoms, and when memory loss is the predominant symptom, it is termed "amnestic MCI" and is frequently seen as a prodromal stage of Alzheimer's disease [11].

**Early-Stage Alzheimer’s disease**

This mild stage, which usually lasts 2 to 4 years, is often when the disease is first diagnosed. In this stage, family and friends may begin to realize that there has been a decline in the patient’s cognitive ability.

The common symptoms include:

- Difficulty retaining new information.
- Difficulty with problem solving or decision making. Patients may start to have trouble managing finances or other instrumental activities of daily living.
- Personality changes. The person may begin to withdraw socially or show lack of motivation.
- Difficulty expressing thoughts.
- Misplacing belongings or getting lost. The patient may have difficulty navigating in familiar surroundings.

**Moderate Alzheimer’s disease**

Lasting 2 to 10 years, this is longest stage of the disease. Patients often experience increased difficulty with memory and may need help with activities of daily living. Symptoms frequently reported during this stage include:

- Increasingly poor judgment and confusion. The patient may begin to confuse family members, lose orientation to time and place, and may begin wandering, making it unsafe for them to be left alone.
- Difficulty completing complex tasks, including many of the instrumental activities of daily living, such as managing finances, grocery shopping, planning, and organization.
- Greater memory loss. Patients may begin to forget details of their personal history.
• Significant personality changes. The person may become withdrawn from social interactions and develop unusually high suspicions of caregivers.

Severe Alzheimer’s disease

Fig.4: Brain condition in final stage of the disease; Comparison between Healthy brain and brain in severe stage of the disease

In this final stage of the disease, cognitive capacity continues to decline and physical ability is severely impacted. This stage can last between 1 and 3 years. Due to the family’s decreasing ability to care for the patient, this stage often results in nursing home or other long term care facility placement. Common symptoms appearing in this stage include:

• Loss of ability to communicate. The patient may still speak short phrases, but are unable to carry on a coherent conversation.
• Reliance on others for personal care, such as eating, bathing, dressing, and toileting. Many patients become incontinent.
• Inability to function physically. The person may be unable to walk or sit independently. Muscles may become rigid and swallowing can eventually be impaired.

Alzheimer Society of Bangladesh (ASB)

Alzheimer Society of Bangladesh (ASB) was born by the strenuous efforts by some dedicated social elites in Thakurgaon district located in North-western part of Bangladesh. But all the credit goes to Md. Azizul Haque who is the incumbent secretary general of ASB first took the lead for its establishment whose father died of Dementia. He asked himself how the disease affects our memory power, thinking, emotion and behavior. He was of the mind it might be a foreign disease the people have less awareness of the disease.
His father Sk. Abdul Malek who was 75 years old suffered for a long time. The doctors could not diagnose him correctly. The doctor opined that he was mentally retarded. The medicines given had been of no help. He passed away on 18th February 2006. Md. Azizul Haque could not accept it realizing that his beloved father would have been died in this way. He got some clues that his father might have suffered with dementia. He took much interest in knowing on Alzheimer’s Disease, bringing its awareness among the common people. By the by he came in close touch with Dr. Md. Nurul Huda, a physician of Thakurgaon, who took much interest on him and advised him to arrange a meeting on Alzheimer Disease. At last Md Azizul Haque with some elites of Thakurgaon invited a meeting on 17th August 2006 in the Community Development Library (CDL) Office Thakurgaon. In this meeting Dr. Md. Momin, Civil Surgeon Thakurgaon was present as chief guest. The meeting was attended by among other many participants who amongst them students, women leaders doctors, nurses, teachers, NGO activists, political leaders, cultural workers, journalists and social workers and thus saw the birth of Alzheimer Society of Bangladesh (ASB) to help the people with Dementia with the spirit of service and love. [12]

MANAGEMENT OF ALZHEIMER’S DISEASE

Currently, there is no cure for Alzheimer’s. But drug and non-drug treatments may help with both cognitive and behavioral symptoms. Researchers are looking for new treatments to alter the course of the disease and improve the quality of life for people with dementia. Current treatments can be divided into pharmaceutical, psychosocial and care giving. Both caregivers and doctors need to remember that no two people with AD are alike. This means that medications may work differently in different people.

Many factors may play a role in the disease, such as:

- Genes
- Lifestyle
- Earlier treatments
- Other illnesses or problems
- The person’s surroundings
- Stage of Alzheimer’s disease.

Medications

Five medications are currently used to treat the cognitive problems of AD: four are acetylcholinesterase inhibitors (tacrine, rivastigmine, galantamine and donepezil) and the other
(memantine) is an NMDA receptor antagonist. The benefit from their use is small. No medication has been clearly shown to delay or halt the progression of the disease.

**Alternative Treatments**

A growing number of herbal remedies, dietary supplements and "medical foods" are promoted as memory enhancers or treatments to delay or prevent Alzheimer’s disease and related dementias. Claims about the safety and effectiveness of these products, however, are based largely on testimonials, tradition and a rather small body of scientific research. The rigorous scientific research required by the U.S. Food and Drug Administration (FDA) for the approval of a prescription drug is not required by law for the marketing of dietary supplements or "medical foods" [13].

- Caprylic acid and coconut oil
- Concerns
- Coenzyme Q10
- Coral calcium
- Ginkgo biloba
- Huperzine A
- Omega-3 fatty acids
- Phosphatidylserine
- Tramiprosate

**Caprylic acid (clinically tested as Ketasyn [AC-1202], marketed as a “medical food” called Axona®) and coconut oil**

Caprylic acid is the active ingredient of Axona, which is marketed as a “medical food.” Caprylic acid is a medium-chain triglyceride (fat) produced by processing coconut oil or palm kernel oil. The body breaks down caprylic acid into substances called “ketone bodies.” The theory behind Axona is that the ketone bodies derived from caprylic acid may provide an alternative energy source for brain cells that have lost their ability to use glucose (sugar) as a result of Alzheimer’s. Glucose is the brain’s chief energy source. Imaging studies show reduced glucose use in brain regions affected by Alzheimer’s.

Axona’s development was preceded by development of the chemically similar Ketasyn (AC-1202). Ketasyn was tested in a Phase II clinical study enrolling 152 volunteers with mild to moderate Alzheimer’s. Most participants were also taking FDA-approved Alzheimer's drugs.
manufacturer of Axona reports that study participants who took Ketasyn performed better on tests of memory and overall function than those who received a placebo (a look-alike, inactive treatment).

**Coenzyme Q10**

Coenzyme Q10, or ubiquinone, is an antioxidant that occurs naturally in the body and is needed for normal cell reactions. This compound has not been studied for its effectiveness in treating Alzheimer’s.

A synthetic version of this compound, called idebenone, was tested for Alzheimer’s disease but did not show any benefit. Little is known about what dosage of coenzyme Q10 is considered safe, and there could be harmful effects if too much is taken.

**Coral calcium**

“Coral” calcium supplements have been heavily marketed as a cure for Alzheimer’s disease, cancer and other serious illnesses. Coral calcium is a form of calcium carbonate claimed to be derived from the shells of formerly living organisms that once made up coral reefs. Coral calcium differs from ordinary calcium supplements only in that it contains traces of some additional minerals incorporated into the shells by the metabolic processes of the animals that formed them. It offers no extraordinary health benefits. Most experts recommend that individuals who need to take a calcium supplement for bone health take a purified preparation marketed by a reputable manufacturer. The Federal Trade Commission (FTC) and the Food and Drug Administration (FDA) have filed formal complaints against the promoters and distributors of coral calcium. The agencies state that they are aware of no competent and reliable scientific evidence supporting the exaggerated health claims and that such unsupported claims are unlawful.

**Ginkgo biloba**

*Ginkgo biloba* is a plant extract containing several compounds that may have positive effects on cells within the brain and the body. *Ginkgo biloba* is thought to have both antioxidant and anti-inflammatory properties, to protect cell membranes and to regulate neurotransmitter function. *Ginkgo* has been used for centuries in traditional Chinese medicine and currently is being used in Europe to alleviate cognitive symptoms associated with a number of neurological conditions.

**Huperzine A**

Huperzine A (pronounced *HOOP*-ur-zeen) is a moss extract that has been used in traditional Chinese medicine for centuries. It has properties similar to those of cholinesterase inhibitors, one
class of FDA-approved Alzheimer's medications. As a result, it is promoted as a treatment for Alzheimer's disease.

The Alzheimer’s Disease Cooperative Study (ADCS) conducted the first large-scale U.S. clinical trial of huperzine A as a treatment for mild to moderate Alzheimer’s disease. Participants taking huperzine A experienced no greater benefit than those taking a placebo.

Because currently available formulations of huperzine A are dietary supplements, they are unregulated and manufactured with no uniform standards. Taking these unregulated preparations could increase the risks of serious side effects, especially if used in combination with FDA-approved Alzheimer's drugs.

**Omega-3 fatty acids**
Omega-3s are a type of polyunsaturated fatty acid (PUFA). Research has linked certain types of omega-3s to a reduced risk of heart disease and stroke.

The U.S. Food and Drug Administration (FDA) permits supplements and foods to display labels with “a qualified health claim” for two omega-3s called docosahexaneoic acid (DHA) and eicosapentaenoic acid (EPA). The labels may state, “Supportive but not conclusive research shows that consumption of EPA and DHA omega-3 fatty acids may reduce the risk of coronary heart disease,” and then list the amount of DHA or EPA in the product. The FDA recommends taking no more than a combined total of 3 grams of DHA or EPA a day, with no more than 2 grams from supplements.

**Concerns about alternative therapies**
Although some of these remedies may be valid candidates for treatments, there are legitimate concerns about using these drugs as an alternative or in addition to physician-prescribed therapy:

**Effectiveness and safety are unknown.** The rigorous scientific research required by the U.S. Food and Drug Administration (FDA) for the approval of a prescription drug is not required by law for the marketing of dietary supplements. The maker of a dietary supplement is not required to provide the FDA with the evidence on which it bases its claims for safety and effectiveness.

**Purity is unknown.** The FDA has no authority over supplement production. It is a manufacturer’s responsibility to develop and enforce its own guidelines for ensuring that its products are safe and contain the ingredients listed on the label in the specified amounts.

**Dietary supplements can have serious interactions with prescribed medications.** No one should take a supplement without first consulting a physician.
DRUGS USED IN ALZHEIMER’S DISEASE

Drugs at a Glance

Several prescription drugs are currently approved by the U.S. Food and Drug Administration (FDA) to treat people who have been diagnosed with Alzheimer's disease. Treating the symptoms of Alzheimer's can provide patients with comfort, dignity, and independence for a longer period of time and can encourage and assist their caregivers as well. [14]

Table 3: List of Drug

<table>
<thead>
<tr>
<th>DRUG NAME</th>
<th>DRUG TYPE AND USE</th>
<th>HOW IT WORKS</th>
<th>COMMON SIDE EFFECTS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aricept® (donepezil)</td>
<td>Cholinesterase inhibitor prescribed to treat symptoms of mild, moderate, and severe Alzheimer's</td>
<td>Prevents the breakdown of acetylcholine in the brain</td>
<td>Nausea, vomiting, diarrhea, muscle cramps, fatigue, weight loss</td>
</tr>
<tr>
<td>Exelon® (rivastigmine)</td>
<td>Cholinesterase inhibitor prescribed to treat symptoms of mild to moderate Alzheimer's (patch is also for severe Alzheimer's)</td>
<td>Prevents the breakdown of acetylcholine and butyrylcholine (a brain chemical similar to acetylcholine) in the brain</td>
<td>Nausea, vomiting, diarrhea, weight loss, decreased appetite, muscle weakness</td>
</tr>
<tr>
<td>Namenda® (memantine)</td>
<td>N-methyl D-aspartate (NMDA) antagonist prescribed to treat symptoms of moderate to severe Alzheimer's</td>
<td>Blocks the toxic effects associated with excess glutamate and regulates glutamate activation</td>
<td>Dizziness, headache, diarrhea, constipation, confusion</td>
</tr>
<tr>
<td>Namzaric® (memantine extended-release and donepezil)</td>
<td>NMDA antagonist and cholinesterase inhibitor prescribed to treat symptoms of moderate to severe Alzheimer’s (for patients stabilized on both memantine and donepezil taken separately)</td>
<td>Blocks the toxic effects associated with excess glutamate and prevents the breakdown of acetylcholine in the brain</td>
<td>Headache, nausea, vomiting, diarrhea, dizziness, decreased appetite</td>
</tr>
<tr>
<td>Razadyne® (galantamine)</td>
<td>Cholinesterase inhibitor prescribed to treat symptoms of mild to moderate Alzheimer's</td>
<td>Prevents the breakdown of acetylcholine and stimulates nicotinic receptors to release more acetylcholine in the brain</td>
<td>Nausea, vomiting, diarrhea, weight loss, decreased appetite</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>DRUG NAME</th>
<th>MANUFACTURER’S RECOMMENDED DOSAGE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aricept® (donepezil)</td>
<td>Tablet*: Initial dose of 5 mg once a day May increase dose to 10 mg/day after 4-6 weeks if well tolerated, then to 23 mg/day after at least 3 months Orally disintegrating tablet*: Same dosage as above 23-mg dose available as brand-name tablet only</td>
</tr>
<tr>
<td>Exelon® (rivastigmine)</td>
<td>Capsule*: Initial dose of 3 mg/day (1.5 mg twice a day) May increase dose to 6 mg/day (3 mg twice a day), 9 mg (4.5 mg twice a day), and 12 mg/day (6 mg twice a day) at minimum 2-week intervals if well tolerated Patch: Initial dose of 4.6 mg once a day; may increase dose to 9.5 mg once a day and 13.3 mg once a day at minimum 4-week intervals if well tolerated Oral solution: Same dosage as capsule</td>
</tr>
<tr>
<td>Drug</td>
<td>Dose and Formulations</td>
</tr>
<tr>
<td>----------------------</td>
<td>---------------------------------------------------------------------------------------</td>
</tr>
</tbody>
</table>
| Namenda® (memantine) | Tablet*: Initial dose of 5 mg once a day  
May increase dose 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 1-week intervals if well tolerated  
Oral solution*: Same dosage as above  
Extended-release capsule: Initial dose of 7 mg once a day; may increase dose to 14 mg/day, 21 mg/day, and 28 mg/day at minimum 1-week intervals if well tolerated |
| Namzaric® (memantine extended-release and donepezil) | Capsule: 28 mg memantine extended-release + 10 mg donepezil once a day  
14 mg memantine extended-release + 10 mg donepezil once a day (for patients with severe renal impairment) |
| Razadyne® (galantamine) | Tablet*: Initial dose of 8 mg/day (4 mg twice a day)  
May increase dose to 16 mg/day (8 mg twice a day) and 24 mg/day (12 mg twice a day) at minimum 4-week intervals if well tolerated  
Oral solution*: Same dosage as above  
Extended-release capsule*: Same dosage as above but taken once a day |

**Limitations of currently available AD drugs** [15]

Currently only five drugs are approved by the Food and drug administration (FDA). Mainly cholinesterase inhibitors such as tacrine, donepezil, rivastigmine, galantamine and NMDA receptor agonist memantine are currently available drugs for the treatment of Alzheimer’s disease (AD). These approved drugs are limited in use due to their acute side effects which are listed in the table below.

Information taken from prescribing information from each drug. Adverse events reported in at least 5% of patient receiving drug at a higher frequency than placebo-treated patient. capsule: 6mg/ day; patch 9.5 mg/day.

**CONCLUSION**

Alzheimer's disease is an irreversible disorder of the brain, which leads to the loss of memory, and overall mental and physical function. Eventually it leads to death. Scientists continue to unravel the complex brain changes involved in the onset and progression of Alzheimer’s disease. It seems likely that damage to the brain starts a decade or more before memory and other cognitive problems become evident. During this preclinical stage of Alzheimer’s disease, people seem to be symptom-free, but toxic changes are taking place in the brain. Abnormal deposits of proteins form amyloid plaques and tau tangles throughout the brain and once-healthy neurons stop functioning, lose connections with other neurons, and die. The damage initially appears to take place in the hippocampus, the part of the brain essential in forming memories. As more neurons
die, additional parts of the brain are affected. By the final stage of Alzheimer’s, damage is widespread, and brain tissue has shrunk significantly.

Although the human and societal cost of Alzheimer’s disease is shocking, there is hope that earlier and better diagnosis, increased knowledge of its natural history with support of the patient and family throughout the disease stages, effective symptomatic drugs, and potentially effective disease modification strategies will have a dramatic impact on the number of persons affected in the future, and the quality of life of persons currently affected. The fast pace of research and development in Alzheimer’s disease is unique in neurological history, and should lead to a better future for aging populations.

Scientists are studying Alzheimer's disease from several different angles. Advances in medical genetics, pharmacologic therapy, and stem cell research hold promise for the Alzheimer's patient. Scientists are testing a number of drugs to see if they prevent Alzheimer's disease, slow the disease, or help reduce behavioral symptoms.

The future builds upon the events and experiences of the past. act’s certainly true of AD research. Our knowledge of AD is advancing rapidly, and we have much to celebrate in our scientific successes.

At the same time, we cannot forget that AD remains an urgent problem for our Nation. e challenge is to continue building on these discoveries so that we can create a brighter future in which the potential of successfully managing AD or even preventing this terrible disease can become a reality.

REFERENCES

2. http://www.slideshare.net/r.yarza/kiki-project


8. Web-MD, 2014


