Introduction
Alzheimer’s disease (AD) is an age-related progressive neurodegenerative disorder and the leading cause of dementia (Querfurth and LaFerla, 2010). It is clinically characterized by progressive deterioration of episodic memory and a global decline of cognitive functions ultimately leading to dependency on custodial care. AD has an insidious onset and it has been estimated that neurodegeneration begins 20–30 years before the clinical manifestations become evident (Mistur et al., 2009). Characteristic neuropathological hallmarks of AD are extracellular accumulation of fibrillar amyloid beta peptides, intracellular neurofibrillary tangles comprised of hyperphosphorylated tau protein and progressive reduction in the number of synapses, dendrites and neurons (Selkoe, 1994; Braak and Braak, 1995).

More women than men have dementia, primarily because women live longer, on average, than men. This longer life expectancy increases the time during which women could develop AD or other dementia. It was estimated that 35.6 million people living with Alzheimer’s disease and other dementias worldwide in 2010, increasing to 65.7 million by 2030 and 115.4 million by 2050. In 2000, India had 3.5 million patients with Alzheimer’s disease as against US, which had 4.5 million patients with Alzheimer’s disease (Upadhyaya et al., 2010). Current data from developing countries suggest that age-adjusted dementia prevalence estimates in 65 years old is 1–3% in India; Alzheimer’s disease accounts for 60% whereas vascular dementia (VaD) accounts for ~30% of the prevalence (Kalaria et al., 2008). World Alzheimer’s report 2010 has highlighted that nearly two-thirds of all people with dementia lived in low and middle income countries, this proportion being set to grow because the sharpest increase in the numbers of people with dementia will be in rapidly developing regions including Latin America, China and India (World Alzheimer report 2010).

The cause or causes of AD are not yet known, most experts agree that AD, like other common chronic conditions, probably develops as a result of multiple factors rather than a single cause. The greatest risk factor for AD is advancing age, but AD is not a normal part of aging. When AD or another dementia is recognized in a person under age 65, these conditions are referred to as “younger onset” or “early-onset” of AD. A small percentage of AD cases, probably less than 1%, are caused by rare genetic variations found in a small number of families worldwide. These variations involve chromosome 21 on the gene for the amyloid precursor protein, chromosome 14 on the gene for the presenilin 1 protein, and chromosome 1 on the gene for presenilin 2. In these inherited forms of AD, the disease tends
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to develop before age 65, sometimes in individuals as young as 30 years. A genetic factor in
late-onset AD (AD developing at age 65 or older) is apolipoprotein E 4 (ApoE 4). ApoE 4 is
one of the three common forms of the ApoE gene, which provides the blueprint for a protein
that carries cholesterol in the bloodstream. Everyone inherits one form of the ApoE gene
from each of his or her parents. Those who inherit one ApoE 4 gene have increased risk of
developing AD. Those who inherit two ApoE 4 genes have an even higher risk. However,
inheriting one or two copies of the gene does not guarantee that the individual will develop
AD. A significant portion of people with mild cognitive impairment (MCI), but not all, will
later develop AD. MCI is a condition in which a person has problems with memory,
language, or another essential cognitive function that are severe enough to be noticeable to
others and show up on cognitive tests, but not severe enough to interfere with daily life.
Studies indicate that as many as 10–20% of people aged 65 and older have MCI. People
whose MCI symptoms cause them enough concern to visit a physician appear to have a
higher risk of developing dementia. It is estimated that as many as 15% of these individuals
progress from MCI to dementia each year. It is unclear what mechanisms put those with MCI
at greater risk for developing AD or other dementia. MCI may, in some cases, represent a
transitional state between normal aging and the earliest symptoms of AD.

Oxidative stress has been implicated to play a crucial role in the pathogenesis of
neurodegenerative disorders including AD (Butterfield and Sultana, 2007, Sultana et al.,
2009). Among all the body organs, the brain is particularly vulnerable to oxidative damage
because of its high utilization of oxygen, increased levels of polyunsaturated fatty acid (that
are readily attacked by free radicals), and relatively high levels of redox transition metal ions;
in addition, the brain has relatively low levels of antioxidants (Butterfield et al., 2002;
Butterfield and Lauderback, 2002). Oxidative stress occurs due to an imbalance in the
prooxidant and antioxidant levels. Reactive oxygen species (ROS) and reactive nitrogen
species (RNS) are highly reactive with biomolecules, including proteins, lipids, carbohydrate,
DNA, and RNA. Oxidative damage to these moieties leads to cellular dysfunctions
(Butterfield et al., 2002; Butterfield and Lauderback, 2002).

AD is also characterized by a chronic inflammatory process around amyloid plaques,
activation of microglia and astrocytes and increased levels of free radicals and
proinflammatory cytokines as well as chemokines. For example, iNOS, IL-1β, IL-6, IL-8 and
TNF-α has been detected in amyloid plaque and plaques surrounding microglia and
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astrocytes (Eikelenboom et al., 2002). In addition to damaging various cell components through oxidation, ROS and RNS can activate redox sensitive transcription factors such as NF-kB and p53. Each of these transcription factors further regulates additional transcription factors through phosphorylation, trigger the generation of proinflammatory molecules including COX-2 resulting in chronic inflammation. Chronic inflammation, in turn, leads to further ROS and RNS generation, consequently amplifying the damaging effects of oxidative stress and chronic inflammation (Finkel and Holbrook, 2000).

It is well documented that AD is associated with oxidative stress. Oxidative stress precipitates apoptotic cellular injury that consists of both nuclear DNA degradation and membrane phosphatidylserine (PS) exposure (Chong et al., 2003; Vincent and Maiese, 1999; Witting et al., 2000). DNA damage leads to the activation of p53 transcription factor which promotes expression of proapoptotic Bcl-2 member’s protein like Bax and suppresses antiapoptotic Bcl-2 and Bcl-XL. Higher expression of Bax leads to increased mitochondrial membrane permeability that leads to release of cytochrome ‘c’ into cytosol, ultimately leads to the formation of apoptosome which consist of Apaf-1, cytochrome ‘c’ and dATP. The apoptosome activates caspase-9 which is initiator caspase and thus is able to mediate the caspase cascade by activating caspase-3 that is the executioner of programme cell death.

Alzheimer's disease is usually diagnosed clinically from the patient history, collateral history from relatives, and clinical observations, based on the presence of characteristic neurological and neuropsychological features and the absence of alternative conditions. Advanced medical imaging with computed tomography (CT) or magnetic resonance imaging (MRI) and with single photon emission computed tomography (SPECT) or positron emission tomography (PET) can be used to help exclude other cerebral pathology or subtypes of dementia. Moreover, it may predict conversion from prodromal stages (mild cognitive impairment) to Alzheimer's disease.

At present, there is no definitive evidence to support that any particular measure is effective in preventing AD (Luchsinger et al., 2007). Global studies of measures to prevent or delay the onset of AD have often produced inconsistent results. However, epidemiological studies have proposed relationships between certain modifiable factors, such as diet, cardiovascular risk, pharmaceutical products, or intellectual activities among others, and a population's
likelihood of developing AD. Only further research, including clinical trials, will reveal whether these factors can help to prevent AD.

Till to date there is no complete cure or treatment for the AD. Blocking cholinesterase-induced hydrolysis of acetylcholine (Ach) and the subsequent increase in Ach concentration in central synapses and the enhancement of cholinergic function—provides the symptomatic improvements observed in patients with probable AD who are treated with cholinesterase inhibitors (Ellis, 2005). Some cholinesterase inhibitors, such as tacrine (Cognex), donepezil (Aricept), rivastigmine (Exelon) or galantamine (Reminyl), enhance the effectiveness of acetylcholine (the chemical messenger found in the neurotransmitter system which coordinates memory and learning) by slowing its breakdown. Unfortunately, these medications only temporally improve the symptoms associated with AD. The effects of the drugs will fade as the deterioration of the brain cells progresses. More recently, memantine (Namenda) was approved by the Food and Drug Administration USA. Memantine blocks the effects of a different chemical, glutamate, which is felt to over-stimulate nerve cells and cause their degeneration. Additionally, doctors may prescribe antidepressants, antipsychotics, anticonvulsants, beta blockers, benzodiazepines, serotonin reuptake inhibitors, and drugs such as Desyrel, BuSpar, and Eldepryl, to control the agitation, psychosis, depressive features, anxious features, apathy and disturbances in sleep and appetite.

Medications and therapies to combat these problems are still in the development of clinical trial stages. For instance, the researches show that vitamin E slow down the progress of some consequences of AD for about 7 months (Doddy et al., 2001), and ginkgo biloba can delay or prevent dementia in older people (Niederhofer, 2010). Scientists are investigating whether estrogen can prevent AD in women with a family history of the disease. Researchers are looking at methods to enhance cerebral metabolism, stabilize membranes, promote neuronal sprouting, decrease inflammation, neurotoxins and excitatory amino acids, as well as alter metabolism of key proteins.