9.1 INTRODUCTION

Alzheimer's disease (AD) is a slow, progressive and a chronic neurodegenerative disease that results in loss of memory, cognition, language skills and drastic behavioural changes which cause 60–70% of dementia. The word 'Alzheimer's' is derived from the German psychiatrist Dr. Alois Alzheimer (1901), who identified the first case of AD in his 50-year-old woman patient, August Deter. He carefully followed her case until she died in 1906 and he then publicly announced the results of his study. Emil Kraepelin described this disease as one which has its own pathological features and also named Alzheimer's disease as presenile dementia, a subtype of senile dementia (Berrios 1990). In 1977 at a conference held for scientists who study AD, he concluded that presenile dementia and senile dementia had the same pathological condition, but the causes for each disease are different. Since then, the term 'Alzheimer's disease' was made official in the medical nomenclature (Amaducci et al. 1986). Epidemiological data indicates a potentially considerable increase in the prevalence of the disease over the next two decades. AD affects up to 5% of people over 65 years, rising to 20% of those over 80 years. AD was estimated to double every 20 years to 66 million and 115 million by 2030 and 2050, respectively (World Alzheimer's Report 2015).

In AD, the neurons in the brain, which produce biochemicals called neurotransmitters, and acetylcholine (ACh) will break their connection with their neighboring neurons and ultimately die. The two main hallmarks by which AD progresses are amyloid plaque, a protein fragment which fails to degrade and accumulates around the neurons, hindering their function and neurofibrillary tangles that are insoluble, twisted tangles of a protein called tau protein that eventually builds inside each affected nerve cell, killing it from within.

The causes of AD include aging, neurofibillary tangles, senile plaque, genetics and others. Each of the above-mentioned causes has its own pathological mechanism by which it progresses to AD (Munoz and Feldman 2000). Aging has always been a risk factor for many diseases, including AD. During the process of cellular respiration, reactive oxygen species (ROS) or free radicals, are produced, which play a crucial role in the development of age-related AD (Smith et al. 1995). Upon upregulation of the activity, an antioxidant enzyme follows with the oxidative damage done to proteins and membrane lipids (Sayre et al. 1997; Smith et al. 1997). A study conducted by researchers at the University of British Columbia indicated an abnormally increased level of iron-handling protein called melanotransferrin, which was observed in the serum of AD patients, thus indicating the mishandling of iron in the neuronal system, and hence it may use as a potential diagnostic tool for the initial study of age-related AD (Kennard et al. 1996).

Inflammation also serves as a mean for AD. The brains of AD patients show evidence of mild active inflammation, including microglial with complementing activation and the presence of inflammatory cytokines (McGeer and McGeer 1995). Significant research has shown that people who take anti-inflammatory agents on a longer basis have a decreased prevalence of AD. On the other hand, women who consumed estrogen were less likely to be diagnosed with AD, and there are supporting studies that showed signs of improvement of AD in women patients (Birge 1997;